The Journal of Bone and Joint Surgery

The Official Publication of
The American Orthopaedic Association
The British Orthopaedic Association
The American Academy of Orthopaedic Surgeons
The Australian Orthopaedic Association
The Canadian Orthopaedic Association
American Society for Surgery of the Hand
New Zealand Orthopaedic Association
South African Orthopaedic Association

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1. Savastano, A. A. et al.: Rhode Island M. J. *41*:425, 1958. 2. Thomson, J. E. M.: Orthopedics *1*:122, 1959. 3. Beegel, P. M.: J. Maine M. A. *49*:86, 1958. 4. Moore, A. T.: J. Bone & Joint Surg. *39A*:811, 1957. 5. Breck, L. W.: Southwestern Med. *35*:207, 1954. 6. Breck, L. W. et al.: Clin. Orthop. No. 12, p. 183, 1958.

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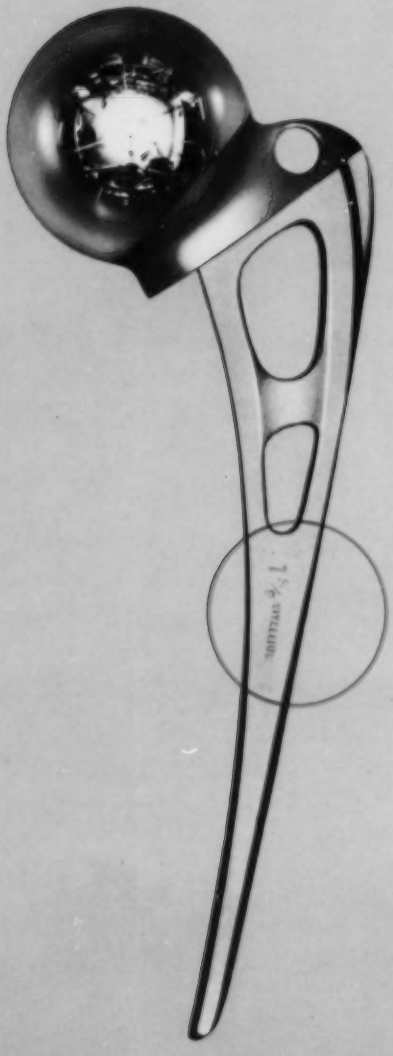

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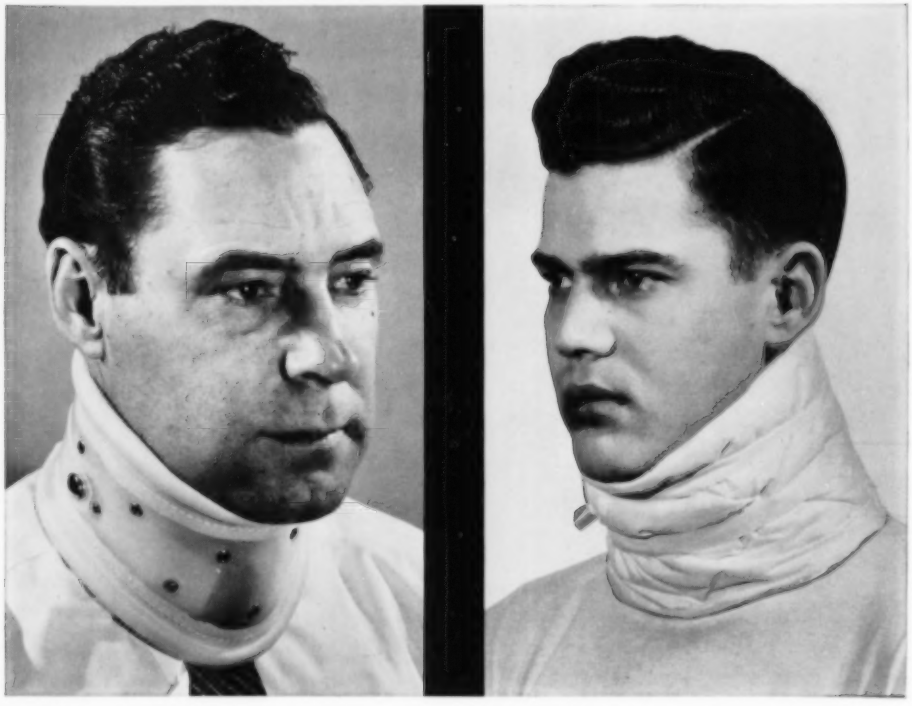

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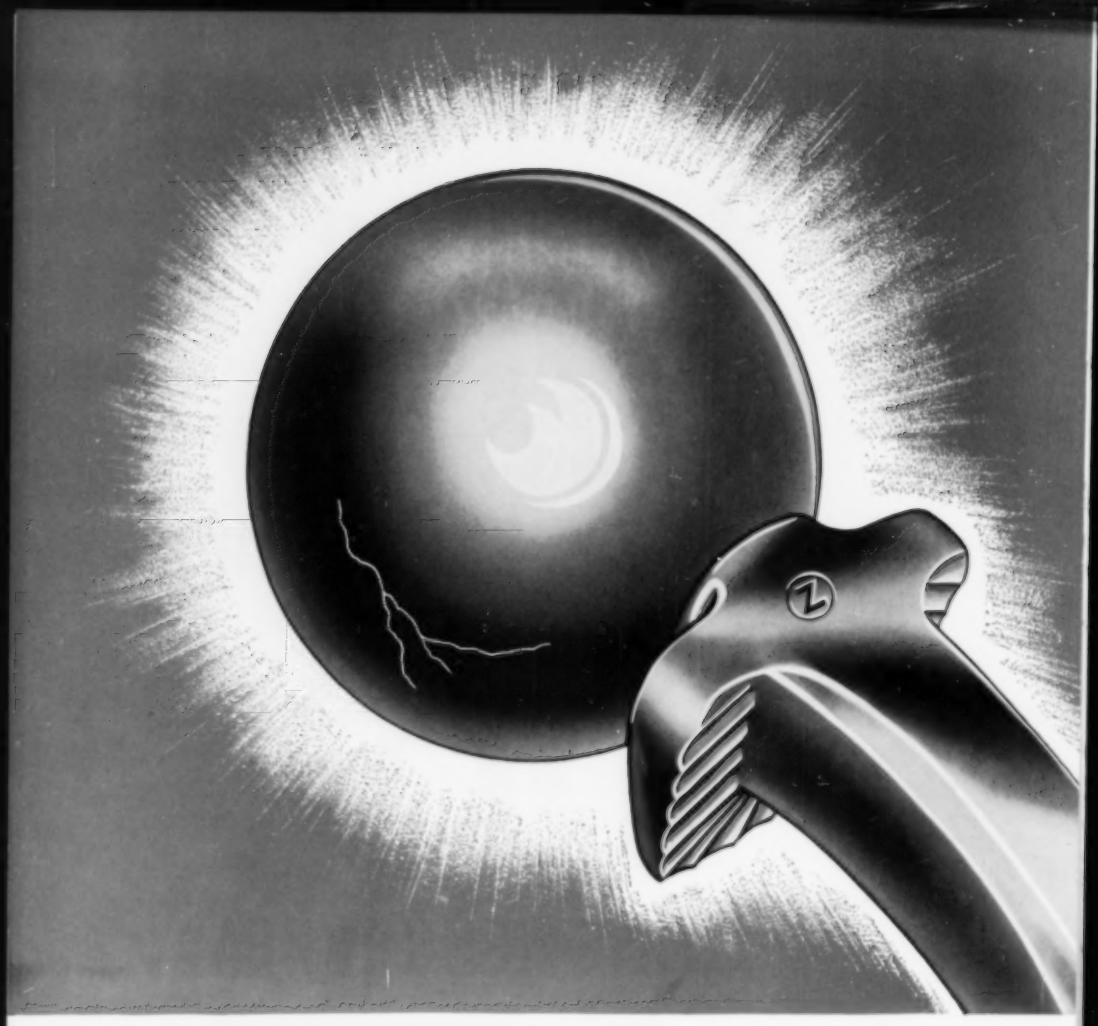

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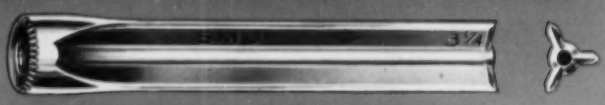

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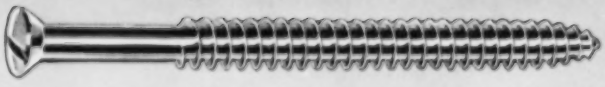

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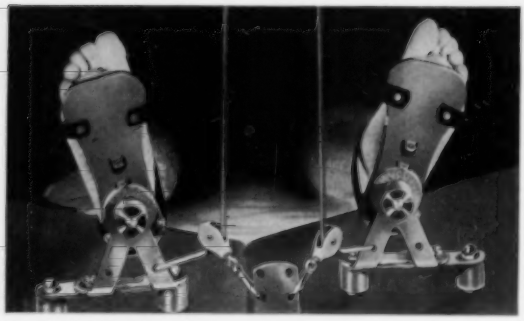

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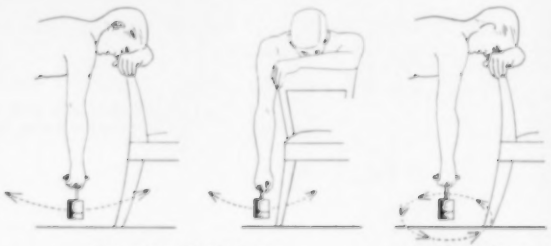

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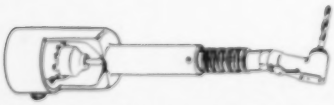

ANGLE DRILL ATTACHMENT

For Luck Bone Saw and Stryker Drill

Designed Especially for the Bankhart Operation

Excellent for bone surgery at difficult angles and in restricted areas requiring use of drills, curetting burrs, slotting burrs. Rigidly constructed, light in weight. Entire unit may be sterilized. Two 1/32 inch diameter High Speed Drills supplied with each attachment.

Easily attached to Jacobs chuck with key.

A hole in the end of the drill bit is provided so that the suture can be threaded and pulled through with the extraction of the drill.

GONIOMETER— Pocket Size Anodized Aluminum

A Pocket Size instrument with calculation flexibility allowing complete angle determination in degrees.

Produced from aluminum, anodized and dyed black, with white-lead-filled dial and indications permitting full penetration of radiation and screening a permanent secondary shadow of measurement on the film for consultations and computations.

BOWEN & COMPANY INC.

P. O. Box 5818
BETHESDA, MARYLAND

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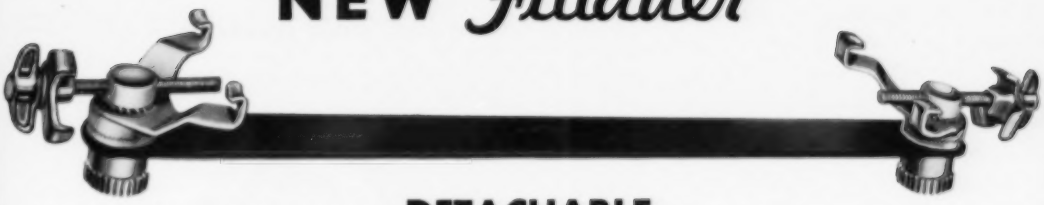
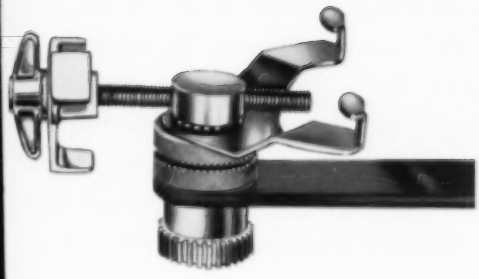
NEW *Fillauer*



DETACHABLE

DENIS BROWNE NIGHT SPLINT



STREAMLINED and IMPROVED

1. Precision die cast Serrated Discs permit 50 positions.
2. New Knurled Screw locks discs in position.
3. New Triple Flange fits club foot shoes or any sole thickness.
4. Streamlined Wing Screw secures flange to shoe sole.
5. Durable, attractive red alumilite color on bars.
6. Lengths 6, 7, 8, 9, 10, 12, 14, 16 and 18 inches. Special lengths to 30 inches at no extra cost.

ORTHOPEDIC SURGEONS recognize the decided advancements this versatile splint affords in treating club feet, positional deformities, tibial torsions, flat feet and congenital hip dislocations. Also applicable for any other deformities that are actively corrected by incessant kicking.

Very little adjustment by surgeon or orthotist is required to fit and position this new Fillauer Detachable Splint.

Pre-walker or larger surgical and club foot shoes work extremely well with the Fillauer Splint. Outgrown shoes may be used by cutting out toes.

Fillauer SURGICAL SUPPLIES, INC.
BOX 1678 CHATTANOOGA, TENN.
ESTABLISHED 1914 *Manufacturers of Orthopedic Supplies*

mind and muscle

in skeletal-muscle disabilities . . .

for whole-patient response in spasm,

Of all muscle relaxants in current use, only meprobamate is supported by hundreds of clinical studies that demonstrate relaxing action on both brain and skeletal musculature. This is why EQUANIL stands as the obvious choice of many physicians concerned with whole-patient response. EQUANIL reduces muscular spasm and tension, aids in the restoration of mobility, speeds rehabilitation, lessens the emotional overlay.[1-6] Its margin of safety is shared by few agents in medical practice.

1. Mitchell, E.H.: M. Ann. District of Columbia *27*:190 (April) 1958. 2. Cooper, C.D., and Epstein, J.H.: Am. J. M. Sc. *235*:448 (April) 1958. 3. Vazuka, F.A.: Neurology *8*:446 (June) 1958. 4. Cobey, M.C.: Am. Surgeon *24*:350 (April) 1958. 5, 6. Wein, A.B.: M. Ann. District of Columbia *27*:346 (July) 1958; Clin. Med. *6*:44 (Jan.) 1959.

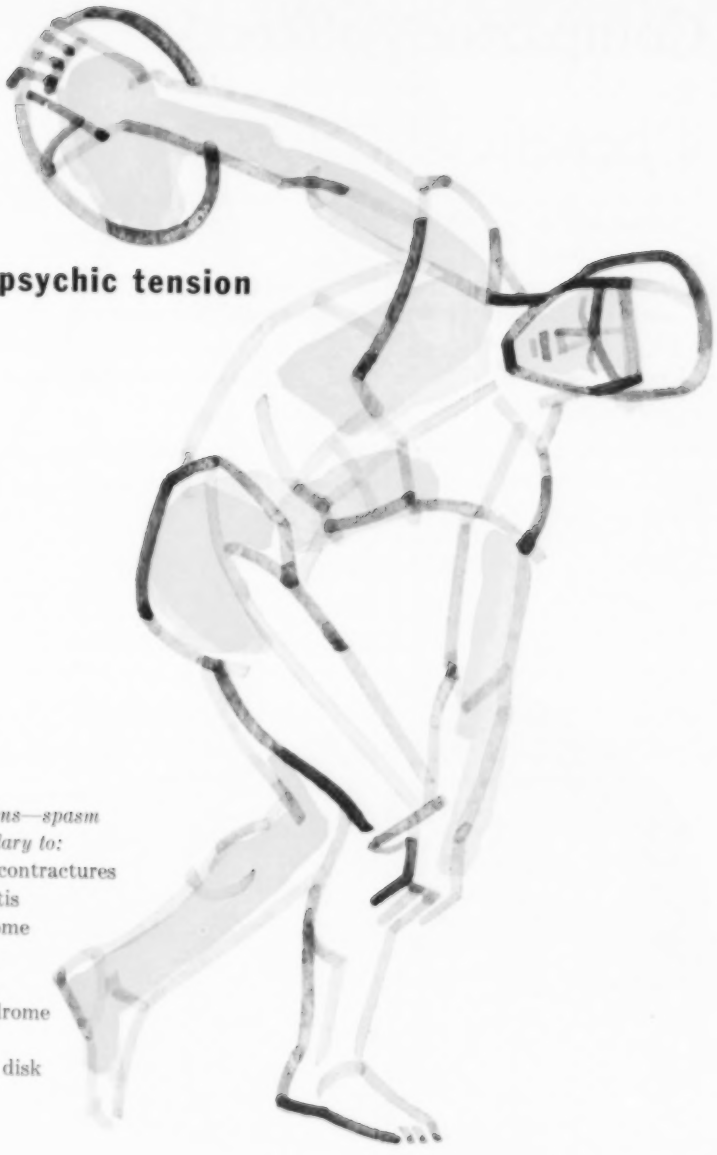
Equanil®

Meprobamate, Wyeth

Wyeth

Philadelphia 1, Pa.

. . . specific central action



rigidity, psychic tension

Partial indications—spasm or tension secondary to:
sprains, strains, contractures
fibrositis, myositis
low-back syndrome
whiplash injury
frozen shoulder
cervical-rib syndrome
herniated intervertebral disk
wryneck
rheumatoid arthritis
rheumatoid or traumatic spondylitis
certain neuromuscular disorders

. . . specific muscular action

In answering advertisements, please mention *The Journal of Bone and Joint Surgery*

in your hospitalized patient

Compazine* offers

4 beneficial effects

- relieves anxiety and tension
- controls nausea
- stops postoperative vomiting
- eases emotional stress that may aggravate pain and other psychosomatic symptoms

Also, hypotension is minimal and infrequent—a particular advantage in surgical patients.

For immediate effect: Ampuls and Multiple dose vials.
Also available: Tablets, Spansule† sustained release capsules, Suppositories and Syrup.

SMITH KLINE & FRENCH LABORATORIES

*T.M. Reg. U.S. Pat. Off. for prochlorperazine, S.K.F.
†T.M. Reg. U.S. Pat. Off.

†surveyor, to non-engineers

he won't be mired for long once he's on

PARAFON*

PARAFLEX® + TYLENOL®

for muscle relaxation *plus* analgesia

prescribe PARAFON in low back pain —sprains—strains—rheumatic pains

Each PARAFON tablet contains:
PARAFLEX® Chlorzoxazone‡ . . 125 mg.
Specific for skeletal muscle spasm
TYLENOL® Acetaminophen . . 300 mg.
The analgesic preferred in musculoskeletal pain

Supplied: Tablets, scored, pink, bottles of 50.

Dosage: Two tablets three or four times a day.

and in arthritis

PARAFON*
with Prednisolone

Each PARAFON WITH PREDNISOLONE tablet contains: PARAFLEX® Chlorzoxazone‡ 125 mg., TYLENOL® Acetaminophen 300 mg., and prednisolone 1.0 mg.

Supplied: Tablets, scored, buff colored, bottles of 36.

Dosage: One to two tablets three or four times a day.

Precautions: The precautions and contraindications that apply to all steroids should be kept in mind when prescribing PARAFON WITH PREDNISOLONE.

McNEIL

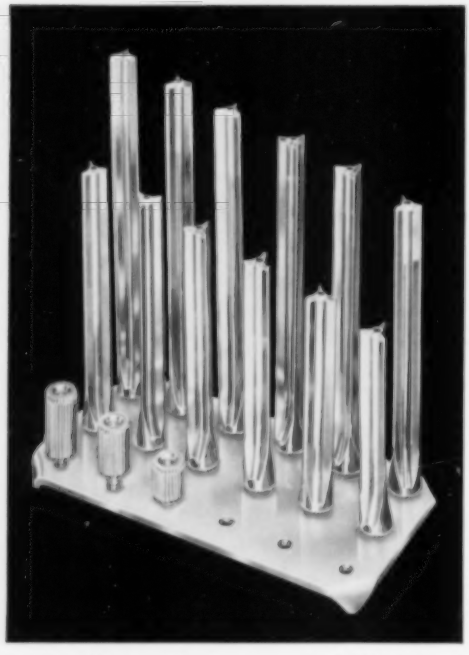
McNeil Laboratories, Inc • Philadelphia 32, Pa.

*Trade-mark ‡U.S. Patent Pending

251A59

NEW ... from Orthopedic

12 SMO SMITH-PETERSEN NAILS
plus 3" x 6" STAINLESS STEEL BASE
IN ONE PACKAGE!



Order now, from your surgical dealer!

Makes Autoclaving Easy!

Pinning a hip? Then you need this Smith-Petersen Nail Assortment . . . 12 nails in a full range of sizes (3" to 6") plus three No. 415 Nail Extensions . . . all on a compact 3" x 6" stainless steel base especially designed for easy autoclaving. Nails are fully cannulated, Style No. 96, to receive 3/32" guide pin and are furnished with deep serrated heads.

COMPLETE PACKAGE (as illustrated) LIST **$100.00**

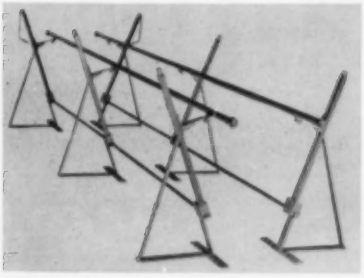
OEC

Plants also in Great Britain and Germany

Orthopedic EQUIPMENT CO. BOURBON · INDIANA

SPLINTS · FRACTURE EQUIPMENT · BONE INSTRUMENTS
SMO INTERNAL BONE APPLIANCES · ORTHOPEDIC SUPPLIES

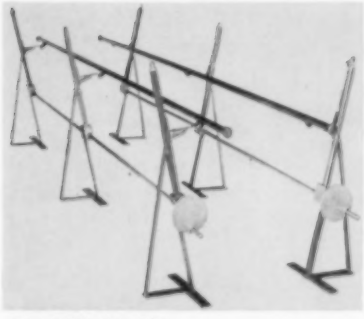
La Berne PARALLEL BARS



MODEL 101 AD

12° angle requires 15" floor space. Each section adjustable in height from 17" to 44". Width adjustment 18" between high and low positions. Operated by folding handle.

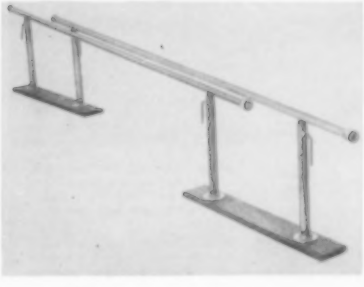
10-12 ft. $295.00
14-16-20 ft. 395.00



Model 301 W—10°

Requires 12" floor space. Each section adjustable in height from 22" to 44". Width adjustment 14" between high and low positions.
Operated by counter-balanced handle.

10-12 feet $295.00
14-16-20 feet $395.00



PORTABLE PARALLEL BARS—No pins to insert adjustable by hand crank through worm and gear.

Bars mounted on platform fitted with 2 sets handrails adjustable in height from 16" to 40" by hand crank. Distance between rails is 24" for upper rails, 19" for lower rails. Platform has slight incline at each end fitted with detachable abduction board. Platform finished in natural wood, handrails finished in Atactic bronze.

Bars require only one person to adjust and may be adjusted by patient from wheelchair. NO PINS to INSERT—worm gears automatically lock bars from moving up or down at any stopped position. Model 175—10 ft. bars $350.00
Upper handrails available in hardwood, no extra cost when specified with order.

ALL PRICES FOB COLUMBIA, S. C.

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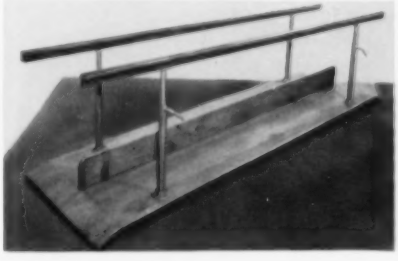


TELESCOPIC MODEL (404)

Mounted on platform. Telescopic height adjustment from 22" to 38". Platform has slight incline at each end, detachable abduction board, platform finished in natural wood. Handrails are hardwood, natural finish. Uprights Mellotone grey.
MODEL 404, 10 feet $175.00
MODEL 410 (as above with 2 pair handrails) $195.00

PORTABLE FOLDING PARALLEL BARS (Model 4400-P)

Telescopic height adjustment from 21" to 38". Requires only 8" floor space when not in use. $98.50

La Berne MANUFACTURING COMPANY

PO Box 5245 Columbia, S. C. Phone SU 7-6162

WALK-OFF

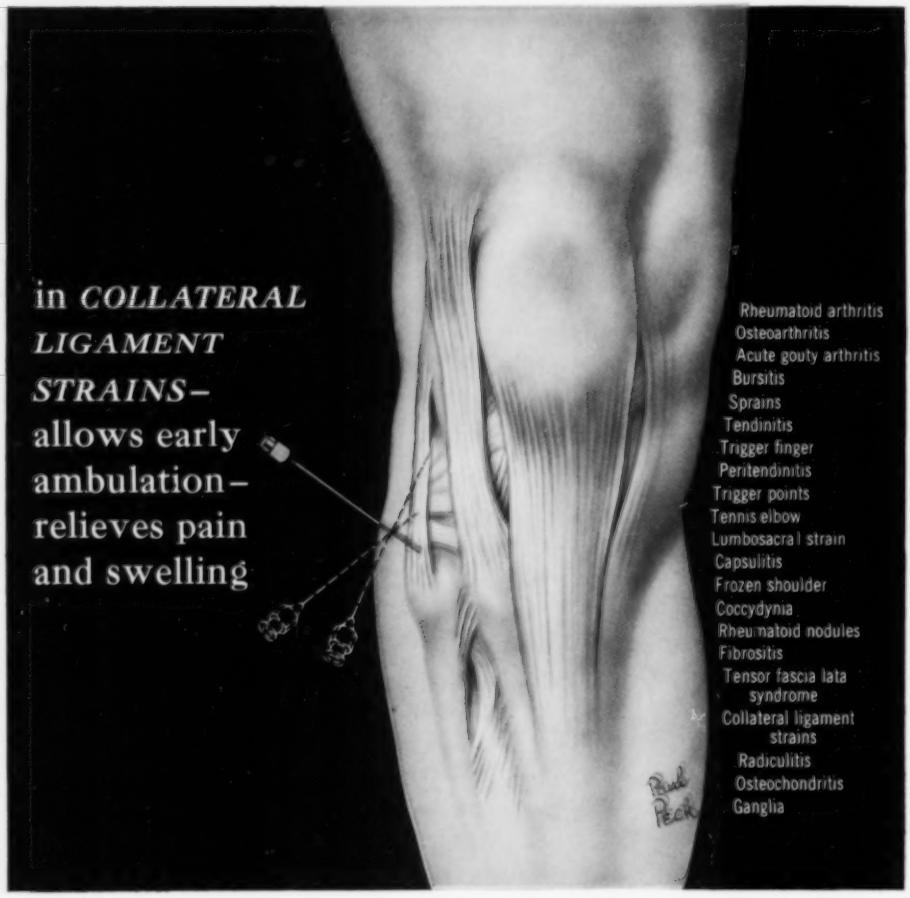
Originators of the "WALK-OFF" Physical Therapy table

In answering advertisements, please mention *The Journal of Bone and Joint Surgery*.

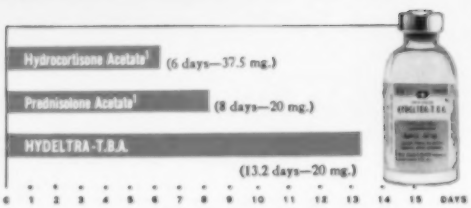
HYDELTRA®-T.B.A.

(Prednisolone tertiary-butylacetate, Merck)

for relief that lasts—longer



Duration of relief exceeds that provided by any other steroid ester


Hydrocortisone Acetate[1]
(6 days—37.5 mg.)
Prednisolone Acetate[1]
(8 days—20 mg.)
HYDELTRA-T.B.A.
(13.2 days—20 mg.)
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 DAYS

Dosage: the usual intra-articular, intra-bursal or soft tissue dose ranges from 20 to 30 mg. depending on location and extent of pathology.

Supplied: Suspension "HYDELTRA"-T.B.A.—20 mg./cc. of prednisolone *tertiary*-butylacetate, in 5-cc. vials.

MERCK SHARP & DOHME
DIVISION OF MERCK & CO., INC.
PHILADELPHIA 1, PA.

After Mastectomy NATURAL APPEARANCE RESTORED, POSTURE AND BODY BALANCE IMPROVED IN A *Spencer*

Spencer's Individual Designing Service enables you to prescribe for the total support requirements of the post-mastectomy patient.

This patient's breast form was created especially for her in the precise size and shape* needed to restore natural appearance.

Her brassiere (with pocket scientifically designed to hold form in place) was made to her *natural* measurements.

A body support was designed individually to . . .

- Support a distressed lumbar curve and reduce lordosis.
- Provide healthful abdominal elevation.
- Help maintain body balance and guard against the postural abnormalities that frequently follow breast surgery.

*Spencer forms may be designed to any shape or thickness to compensate for the most radical surgical procedures.

Spencer's Individual Designing Service is available to you through Corsetieres specially trained to help you help your patients.

SPENCER®

individually designed supports for women, men and children

SPENCER, INCORPORATED
19 Ellsworth Ave., New Haven 7, Conn.
Canada: Spencer, Ltd., Rock Island, Quebec
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☐ Send me the name of the nearest Spencer Corsetiere.

☐ I would like an office demonstration of Spencer benefits and features.

Name _____

Address _____

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7-59

New SABEL'S EQUINO-VARUS TRADE MARK REG. U.S. PAT. OFF.

CONVERTIBLE
BRACE SHOE

Sabel introduces a newer, cleaner, simpler brace and shoe combination that simplifies the prescribing of corrective footwear by the doctor, for all time. The shoe features the Roto-Lok insert, a built-in serrated disc that is attached to the sturdy steel shank . . . no abrasive or scratching action. It's the perfect night . . . and day shoe . . . with or without the brace. Saves an extra pair of shoes.

. . . with a

BUILT-IN ROTO-LOK INSERT

The setting of the splint on the shoe is made easy by a protractor that is stamped on the sole of each shoe.

EMBOSSED PROTRACTOR STAMPED ON SOLES

The brace has a matching serrated disc and smooth knurled nut that screws into the disc in the sole of the shoe, holding it firmly in the position the doctor desires.

This smoother brace has no protruding arms or edges to cut or tear bed clothing.

R. J. POTVIN SHOE CO., BROCKTON, MASS.

SUSAN'S IDEA TOOK THE WAIT OUT OF THE WAITING ROOM

She kept complaining about my old x-ray machine — said she could accomplish more if only she had that new G-E unit I'd talked about. She'd have fewer retakes too — most of them were caused by the long exposures necessary with low power.

From the day my new Patrician combination arrived I'm sure Susan felt her persistence had turned the trick. (And you know — she *is* working faster today!)

Patrician speeds x-ray examinations

. . . and for such modest cost

You'll find your work load lighter with Patrician's big-table convenience. Best news is 200-ma, 100-kvp power, electronically timed. Self-tending reciprocatic Bucky. Finger-tip control of fluoroscopic screen or optional spot-film device. Angulation to 15° Trendelenburg. Automatic Bucky-slot closures for x-ray safety. Ask your G-E x-ray representative for full details. Or clip coupon for a copy of our fully illustrated catalog.

X-RAY DEPARTMENT
GENERAL ELECTRIC CO.
Milwaukee 1, Wisconsin, Rm. CF-71

- ☐ Please send me your 16-page PATRICIAN bulletin
- ☐ Facts about deferred payment
- ☐ MAXISERVICE ® rental

Name .....

Address .....

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Progress Is Our Most Important Product

GENERAL ELECTRIC

". . . he runs and plays again!"

Hanger Prosthetic Appliances have brightened the present and the future for many amputees. For example, Weaver Nolt says: "My son, Lloyd, was a pathetic figure in a big hospital bed after his legs were amputated because of an accident. Today it's a big and wonderful world again as he gets along so wonderfully on his Hanger Legs. He walks without any help, and runs and pushes his wagon all over the farm. That other day is just a hazy memory, and we are so pleased things are so different than we expected."

Hanger PROSTHETIC

AVAILABLE AT AUTHORIZED FACILITIES IN THE FOLLOWING CITIES:

Eastern Region:

BALTIMORE 1, MD.
BOSTON 15, MASS.
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NEW YORK 11, N. Y.
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RICHMOND 19, VA.
ROANOKE 12, VA.
WASHINGTON 13, D. C.

Central Region:

COLUMBUS 8, OHIO
JOHNSTOWN, PA.
PITTSBURGH 1, PA.

Southeastern Region:

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BIRMINGHAM 1, ALA.
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JACKSONVILLE, FLA.
MIAMI 37, FLA.
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MONTGOMERY, ALA.
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CINCINNATI 29, OHIO
DALLAS 1, TEXAS
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FORT WAYNE, IND.
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WICHITA, KANS.

In answering advertisements, please mention *The Journal of Bone and Joint Surgery.*

How many patients today complained about pain?

non-narcotic—pain relief equivalent to that of codeine

well tolerated in both acute and prolonged use

wide range of indications—general practice and the specialties

analgesia plus anti-inflammatory action

Supplied: Tablets, bottles of 48. Each tablet contains 75 mg. of ethoheptazine citrate and 325 mg. (5 grains) of acetylsalicylic acid.

remember

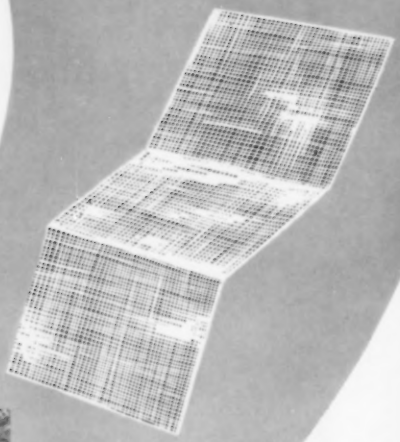
PAIN

Zactirin®

Ethoheptazine Citrate with Acetylsalicylic Acid, Wyeth

Wyeth
Philadelphia 1, Pa.

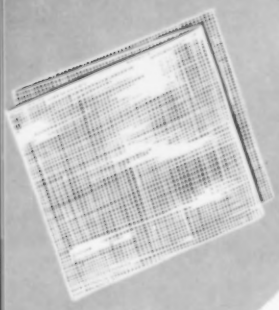
The NEW DUAL PURPOSE



VASELINE
PETROLATUM GAUZE U. S. P.
STERILE
3 x 3 PAD, opens to 3 x 9 INCHES
UNOPENED ENVELOPE MAY BE AUTOCLAVED OR COLD-SOAKED
TEAR HERE

3″ x 9″ STRIP

Shorter length ends waste on small area wounds. New Z-fold insures perfect graft takes. Guaranteed sterile at time of use.



3″ x 3″ PAD

Three-ply, fine-mesh gauze, lightly impregnated — for use in physician's office, industrial medical department, first aid.

6th SIZE of VASELINE® PETROLATUM GAUZE

Now supplied in:

1/2″x 72″	3″x 18″
1″x 36″	3″x 36″
3″x 3″ / 3″x 9″	6″x 36″

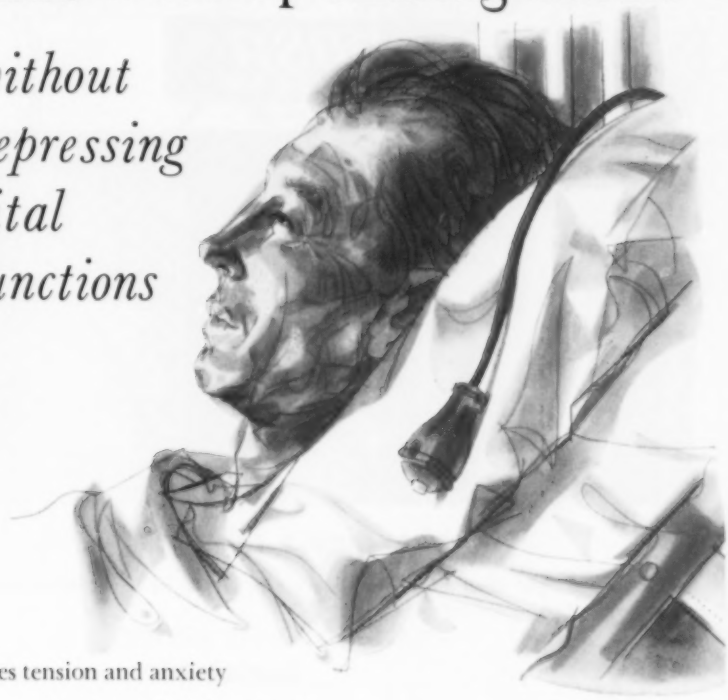
Sole Maker:

CHESEBROUGH-POND'S INC.
Professional Products Division
New York 17, N. Y.

VASELINE *is a registered trademark of Chesebrough-Pond's Inc.*

Relieves Pre-op Apprehension Aids Post-op Management

without depressing vital functions



- relieves tension and anxiety
- helps provide "a good night's sleep"
- facilitates early ambulation
- relaxes skeletal muscle

Miltown has no adverse effects on cardiovascular, respiratory or gastrointestinal functions and causes no depressive "hang-over"

Miltown®
meprobamate (Wallace)

Miltown is available in 400 mg. scored and 200 mg. sugar-coated tablets; bottles of 50.

WALLACE LABORATORIES
New Brunswick, N. J.

Reprints available on request:

1. Kern, E. C.: Preoperative sedation with meprobamate (Miltown) in surgery of eye, ear, nose and throat. Eye, Ear, Nose & Throat Month. 36:408, July 1957.
2. Lamphier, T. A.: The value of meprobamate preoperatively. Maryland M.J. 7:627, Nov. 1958.
3. Lamphier, T. A.: Meprobamate—an important adjunct to post-operative surgical care. J. Maine M.A. 48:357, Oct. 1957.
4. Lamphier, T. A.: The role of meprobamate in postoperative surgical care. Ann. New York Acad. Sc. 67:810, May 9, 1957.
5. Rushia, E. L.: Preliminary report on the use of meprobamate for pre-anesthetic sedation. J.M.A. Georgia 46:93, March 1957.
6. Sadove, M. S. and Schiffrin, M. J.: Tranquilizers in surgery, in Surg. Clin. North America, Saunders, Philadelphia, Feb. 1959, pp. 231-244.

CM-8879

Lock the Bone with Dynamics

SUMMARY OF BASIC TECHNICS*

The RUSH® MEDULLARY PIN

PAT. NO. 2,579,968

COMPLEX ANKLE FRACTURE

The pins here were straight when driven. Curves were produced by intraosseous pressures to give stable fixation without need for external splinting.

SINGLE PIN

Indicated when both fragments have substantial length.

STRAIGHT PIN: Areas indicated in black.

CURVED PIN: Shafts of humerus, femur and tibia. Resists muscle angulating force by three point pressure.

Humerus Shaft

Femur Shaft

Tibia Shaft

* Rush, Leslie V.: Fracture Problems, Mississippi Doctor, Vol. 36, No. 5:115–118, Oct., 1958.

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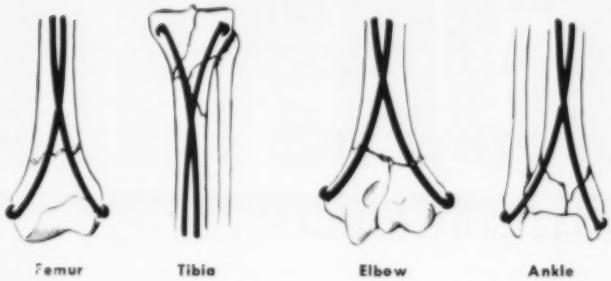
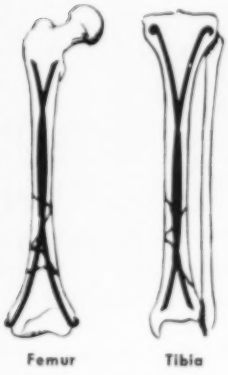
DOUBLE PINS

SIMILAR ANATOMIC AREAS

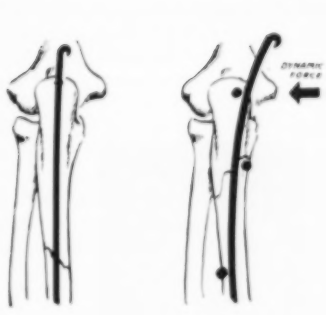


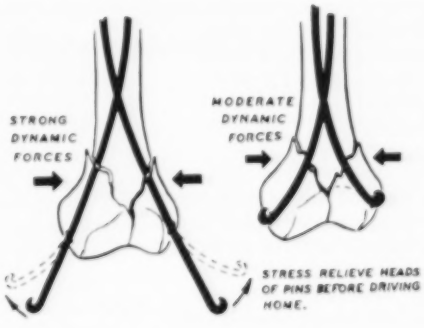


COMMINUTED FRACTURES

Similar Problems: Closed pinning best. Relatively good bone length can be maintained.

STRESS RELIEF OF HEADS





Pin inserted through end of bone exerts no dynamic force. Stress relief not necessary.

Pin inserted through side of bone is forced into curve. Result: Strong dynamic force with three point pressure. Head of pin can sink into bone unless stress relieved.

WRITE FOR INFORMATION
Exclusive Manufacturers of
RUSH MEDULLARY PINS AND INSTRUMENTS
Publishers of
"ATLAS OF RUSH PIN TECHNICS"
by LESLIE V. RUSH, M. D.

THE BERIVON *Company*
MERIDIAN, MISS.

In answering advertisements, please mention *The Journal of Bone and Joint Surgery.*

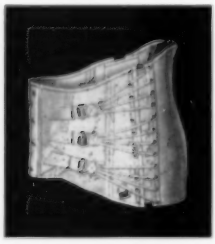
A fitting service you can prescribe, with confidence . . .

Your Truform anatomical supports dealer is an expertly trained and skilled technician.

When you prescribe Truform you can count on him, and his staff, to select and fit the right item to provide effective support or effective fixation.

Training at factory schools and in refresher courses assures this: accurate fitting of quality Truform supports, to provide the maximum therapeutic effect consistent with comfort.

Truform anatomical supports are available only from the Ethical Appliance Dealer.



Three pull straps control traction in this Model 430-EHS lumbo-sacral support . . . two rigid steels

TRUFORM *anatomical supports*

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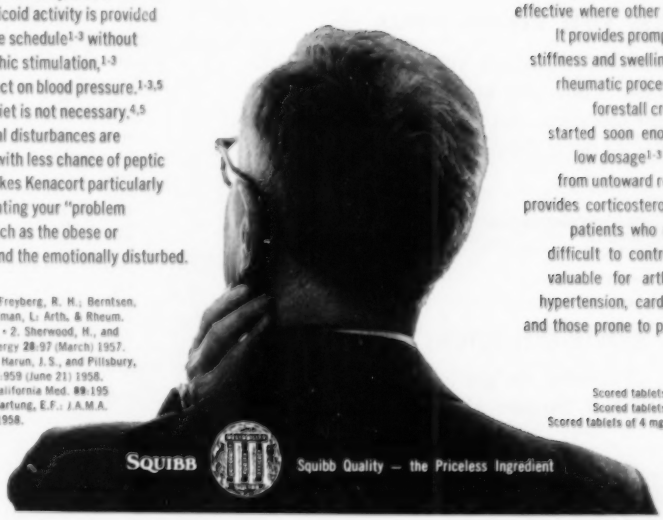

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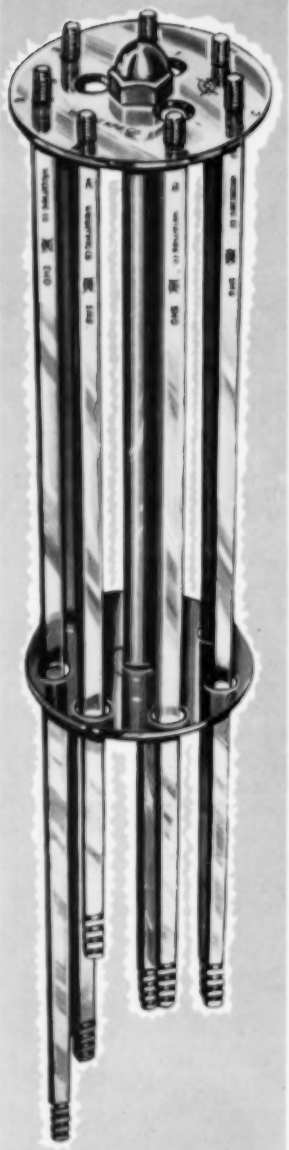

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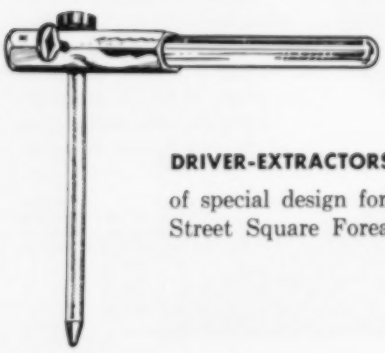

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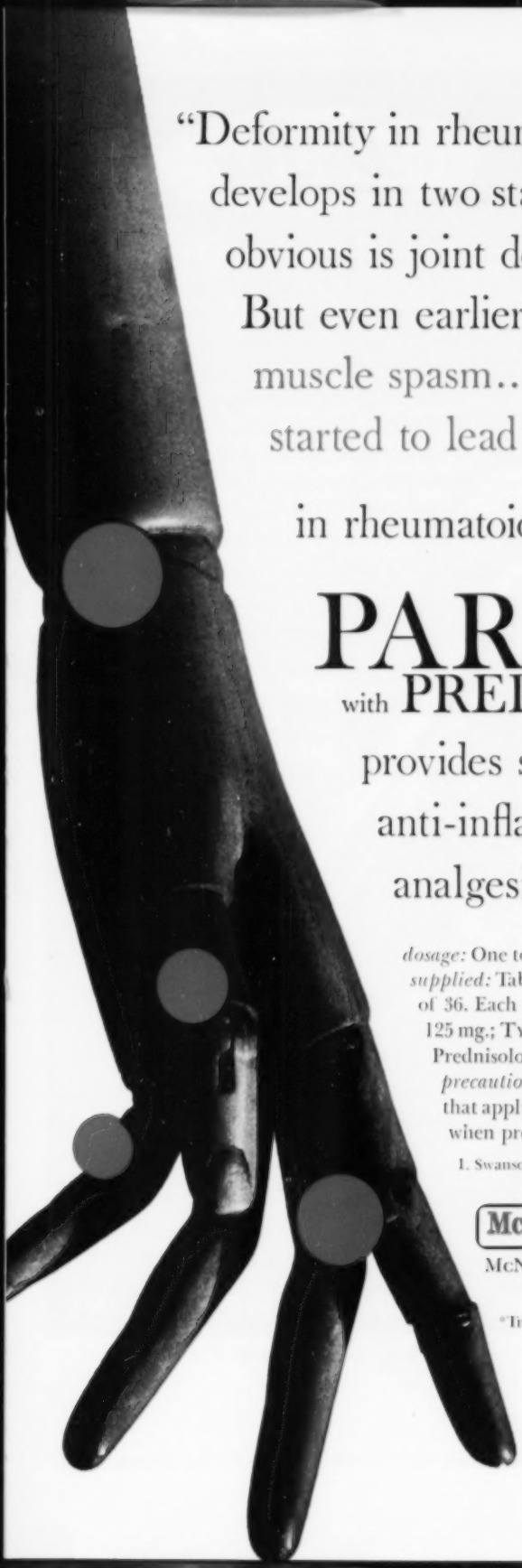

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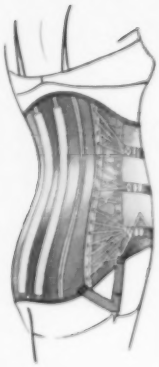

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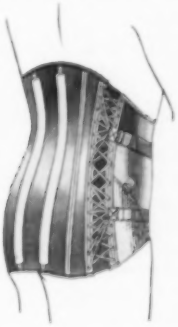

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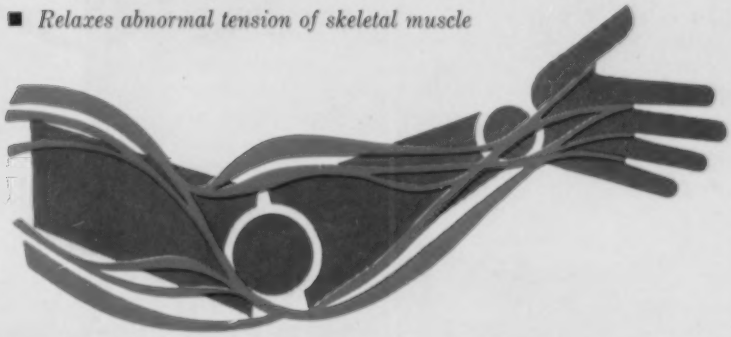

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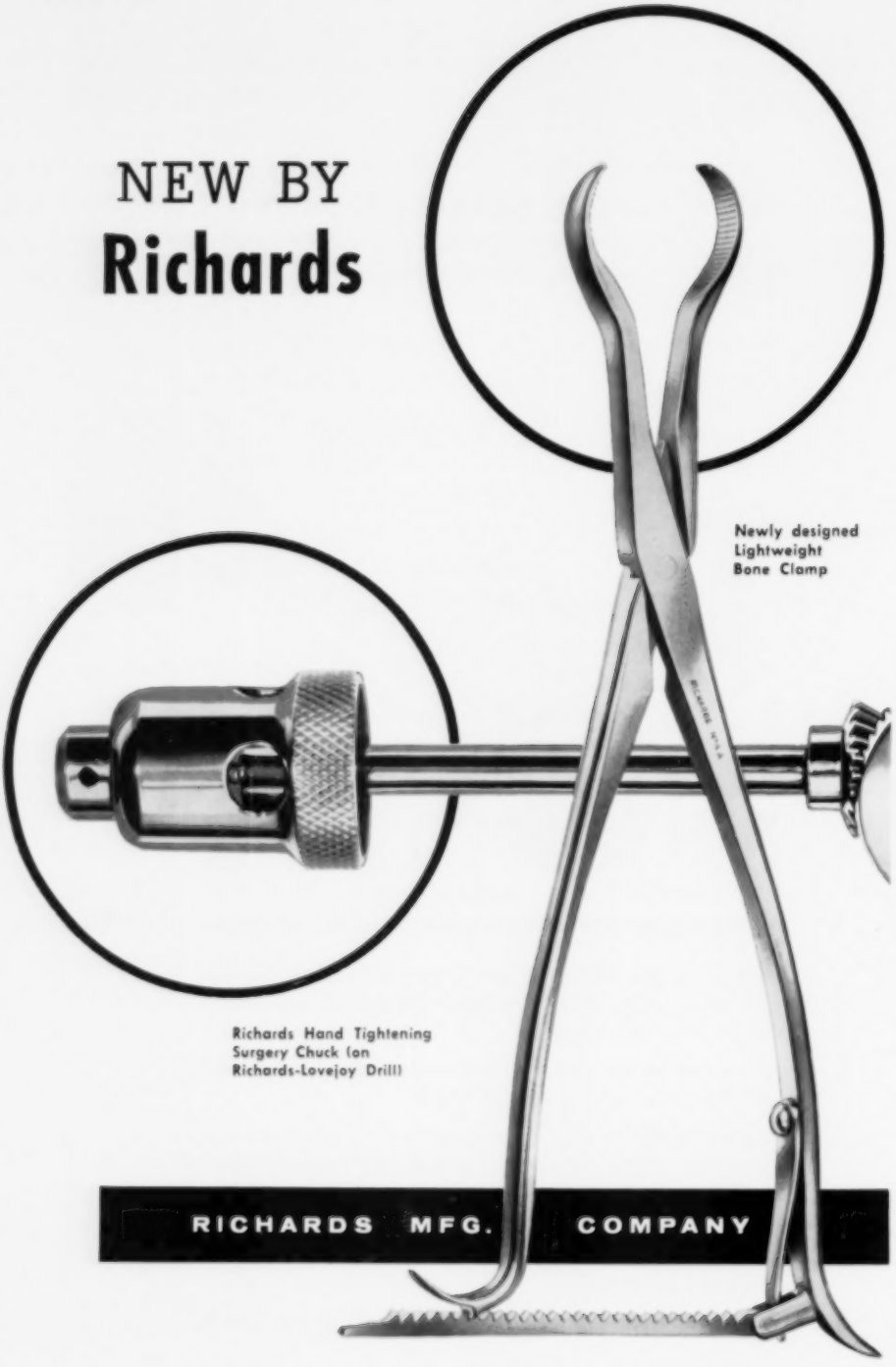

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The Journal of
Bone and Joint Surgery
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Bone and Joint Changes Following Burns

A Roentgenographic Study—Preliminary Report *

BY E. BURKE EVANS, M.D., AND JAMES R. SMITH, M.D., GALVESTON, TEXAS

From the Department of Surgery, Divisions of Orthopaedic and Plastic Surgery, University of Texas, Medical Branch, Galveston

Bone and joint changes occurring as a result of severe burns are of clinical interest because of their significance in the rehabilitation phase of treatment, and of academic interest from the standpoint of pathogenesis.

In the literature there has been modest reference to skeletal change as a complication of burns. Articles have dealt separately with osteoporosis [2,8] or ectopic bone [4], with occasional allusion to other changes. Only recently has there appeared, in the German literature, an article which covers to some extent the calcific changes [5,6]. Artz and Reiss, in their book on the treatment of burns, give some suggestion of the variety of changes, but without elaboration.

We have been impressed not so much by the frequency of occurrence of skeletal changes, as by their variety and by their importance as deterrents to remobilization. Therefore, in January of 1957 there was begun by the Divisions of Orthopaedic and Plastic Surgery a cooperative study aimed principally at obtaining routine roentgenograms at regular intervals of all severely burned patients treated at John Sealy Hospital on an in-patient basis, beginning as soon as practicable after injury. Blood and urine chemistry determinations, blood cultures, and gross and histological examination of autopsy, surgical, and biopsy specimens were undertaken as part of the study. In addition to this, the available roentgenograms of all burned patients treated from 1950 through 1956 were reviewed.

It is apparent that several years will be required for the accumulation of adequate data from laboratory sources. Roentgenographic studies have, however, been rewarding, and from the serial roentgenograms and those made prior to 1957 we have collected twenty cases (Table I) which demonstrate skeletal changes other than osteoporosis—osteoporosis being, in one degree or another, a rather consistent finding in all severely burned patients. The total number of burned patients treated as in-patients from January 1950 to the time of writing is 950.

From the study of the twenty cases we have determined the changes, which include, in addition to osteoporosis, periosteal new-bone formation, pericapsular calcification, osteophyte formation, heterotopic para-articular ossification, and progressive joint destruction and ankylosis. This classification is arbitrary, based on the way the alterations appeared to us roentgenographically as discrete and independent findings; there will doubtless be revisions as we gain more experience.

Serial roentgenography has in some instances shown the progression of change from normal bone to advanced skeletal involvement; in others it has disclosed subtle changes which would otherwise have gone unrecognized for lack of clinical indication for investigation. Sufficient cases have not yet been observed from the

* This investigation was supported in part by Research Grant No. A 848 (C-1, 2, and 3) from the National Institute of Arthritis and Metabolic Diseases, United States Public Health Service.

TABLE I

SKELETAL ALTERATIONS AFTER BURNS

Case	Patient	Sex	Age at Burn (*Years*)	Extent and Distribution of Third-Degree Burn	Time after the Burn at Which There Was Roentgenographic Recognition (*Months*)	Type of Alteration	Proximity to Burn	Clinical Evidence at Time of Recognition
1	M.C.	F	3	20 per cent; lower part of abdomen, left hand, buttocks, thighs	9	Intra-articular ankylosis, both hips	Adjacent	Limitation of motion
2	J.C.	M	7	20 per cent; right lower extremity	6	Fibrous ankylosis, right knee	Adjacent	Limitation of motion, knee and ankle
						Intra-articular ankylosis, right ankle and right subtalar joint	Adjacent	
3	E.S.	F	9	50 per cent; abdomen, back, arms, left thigh, upper part of right thigh	8	Periosteal new-bone formation, ribs	Adjacent	Limitation of motion, both hips and right elbow
						Intra-articular ankylosis, left hip	Adjacent	Fusiform swelling and limitation of motion, right ankle
						Heterotopic-bone bridging, right hip	Adjacent	
						Extra-articular ankylosis, left elbow	Non-adjacent	
						Intra-articular ankylosis, right ankle and right subtalar joint	Non-adjacent	
4	J.E.	F	25	50 per cent; abdomen, back, arms, legs	18	Osteophyte formation, right elbow	Adjacent	Fusiform swelling
5	I.W.	M	10	30 per cent; back and left upper extremity	11	Pericapsular calcification, left elbow	Adjacent	Limitation of motion
6	L.C.	F	39	17 per cent; upper part of back and both arms	15	Osteophyte formation, both elbows, with ankylosis, left elbow	Adjacent	Limitation of motion
7	R.B.	M	16	40 per cent; chest, back, right arm, left forearm, left hand	20	Osteophyte formation, right elbow	Adjacent	Limitation of motion, right elbow
						Irregular ossification, both iliac crests	Adjacent	Granulating area over iliac crests
8	R.D.	M	26	18 per cent; both arms, both thighs	6	Osteophyte formation, left elbow	Adjacent	Limitation of motion
9	C.H.	M	9	30 per cent; both lower extremities	6	Periosteal new-bone formation, both tibiae and fibulae	Adjacent	Diffuse swelling, legs and ankles, with open granulations
						Intra-articular ankylosis, both ankles	Adjacent	
10	L.S.	F	4	40 per cent; back, chest, abdomen, left arm, both thighs	5*	Intra-articular ankylosis, left wrist	Direct	Bones of wrist exposed
						Intra-articular ankylosis, left elbow	Adjacent	Fusiform swelling and limitation of motion, elbow

11	B.E.	M	30	30 per cent; chest, back, both upper extremities	7	Pericapsular calcification, both shoulders	Non-adjacent	No evidence at shoulders
						Osteophyte formation, both elbows	Adjacent	Limitation of motion, both elbows
12	C.F.	M	38	40 per cent; chest, abdomen, right upper extremity, right thigh	6	Osteophyte formation, right elbow	Adjacent	Fusiform swelling and limitation of motion
13	S.McK.	F	48	10 per cent; upper part of back, left buttock	10	Heterotopic-bone bridge, right shoulder	Non-adjacent	Limitation of motion, both shoulders and right elbow
						Pericapsular calcification, left shoulder	Adjacent	
						Osteophyte formation with ankylosis, right elbow	Non-adjacent	
14	P.D.	F	7	40 per cent; neck, chest, abdomen, arms, left forearm, thighs	3*	Intra-articular ankylosis, right elbow	Non-adjacent	Fusiform swelling, heat, tenderness, limitation of motion, both elbows
						Pericapsular calcification, left elbow	Adjacent	
15	R.G.	M	5	40 per cent; abdomen, back, left arm, both thighs	3*	Heterotopic para-articular ossification, both hips	Adjacent	Hips underlay granulating area
						Heterotopic ossification, right elbow	Non-adjacent	Limitation of pronation and supination, right forearm
16	B.P.	M	9	55 per cent; face, neck, chest, back, abdomen, groin, both arms, both thighs	2*	Pericapsular calcification, right elbow	Adjacent	Fusiform swelling, heat, tenderness, limitation of motion
17	J.W.	M	8	16 per cent; face, neck, chest, right upper extremity	5*	Heterotopic bone, right elbow	Adjacent	Limitation of motion
18	S.R.	F	10	18 per cent; face, neck, chest, back, right upper extremity	5*	Pericapsular calcification, right elbow, with adjacent periosteal elevation	Adjacent	Fusiform swelling, persistent granulations, limitation of motion
19	P.W.	F	12	60 per cent; chest, back, both upper extremities, buttocks, thighs	2*	Pericapsular calcification, right shoulder	Adjacent	Fusiform swelling, heat, tenderness, limitation of motion, shoulder and elbow
						Intra-articular destruction, right elbow, with subluxation	Adjacent	
20	J.M.	M	18	10 per cent; both legs, right foot, lower part of left thigh	2*	Periosteal new-bone formation, right tibia and fibula	Adjacent	Overlying granulations and exposed bone
						Tangential sequestration, right tibia	Direct	

* The time of recognition in only these cases is known to approximate the actual time of onset.

acute stages to enable us accurately to anticipate change or to predict time of onset.

This preliminary report concerns the roentgenographic findings in selected patients with thermal burns with osteoporosis alone and in twenty patients with skeletal changes in addition to osteoporosis.

These skeletal alterations fall logically into three groups:

1. Alterations limited to bone: osteoporosis and periosteal new-bone formation;

2. Alterations involving pericapsular structures: pericapsular calcification, osteophytes, and heterotopic para-articular ossification;

3. Alterations involving the joint proper: progressive articular destruction and ankylosis.

Alterations Limited to Bone

Osteoporosis

Some degree of bone atrophy or osteoporosis is common to all severely burned individuals. Owens called attention to the importance of osteoporosis as a complication and emphasized the role of hyperaemia in the deossification process. Artz and Reiss, in a paragraph dealing with the subject, state that there are two factors in burns that are known to be associated with osteoporosis: (1) increased adrenocortical activity, and (2) prolonged immobilization. Colson, Stagnara, and Hounot analyzed burns with osteoporosis and classified them as (1) localized osteoporosis in deep or infected burns of the extremities, (2) diffuse painful osteoporosis, and (3) latent osteoporosis in burned individuals with scarring. These writers did not discount local factors, but they emphasized the role of reflex vasomotor phenomena in the production of osteoporosis.

We have not undertaken, as yet, a correlated study of the various types of bone atrophy, but we have made certain observations in the cases thus far reviewed.

Bone atrophy may appear as a local phenomenon in

FIG. 1

FIG. 2

Fig. 1: Case 7. Although there is generalized osteoporosis, the bones of the right upper extremity reflect the greater severity of the burn on that side, four and one-half years after the acute burn. This patient also showed irregular ossification of the iliac crests and osteophytes of the right elbow.

Fig. 2: Three months after injury the roentgenogram shows marked atrophy of the bone limited to the distal radius and ulna, the wrist, and the hand of the extremity with a minimal third-degree burn. There was no other skeletal alteration.

patients with burns limited principally to an extremity, or it may be generalized in individuals who are more diffusely involved, who must remain immobile for a long period of time.

In the locally burned individual, the atrophy may appear in all bones of the extremity, and it is presumed that this type of atrophy is secondary to the local hyperaemia and the chronic inflammatory state which abides with granulating surfaces and grafting procedures and also to immobility of the part. The fact that the degree of atrophy varies with the extent of local involvement is illustrated in Figure 1.

Local atrophy, on the other hand, may be very limited in extent but of great intensity and of abrupt onset in an extremity with only small areas of third-degree burn, as shown in Figure 2, and it is presumed that there is in these instances a vasomotor reflex phenomenon which contributes to the local hyperaemic state. The extremity shown in Figure 2 appeared clinically to be the same as Sudeck's atrophy but the characteristic pain was lacking.

Generalized atrophy is slowly progressive in individuals with an extensive burn. There may be diffuse atrophy of one degree and a more intense atrophy in the more severely burned areas. Diffuse symmetrical involvement is uncommon. The time after the acute injury at which atrophy is easily detectable on a roentgenogram varies considerably among patients. Figures 8-B and 8-F give some indication of the degree of diffuse atrophy in Case 3 nine months after the acute burn.

Atrophy of the generalized type is thought to be secondary to local hyperaemia, chronic local inflammatory processes, and prolonged inactivity. We accept the less tangible factors of malnutrition and protein insufficiency and of adrenocortical hyperactivity. Artz and Reiss stated that the extent of osteoporosis is related to the severity of injury and the length of immobilization, and that the more severe burns are also associated with more intense and more prolonged hyperadrenocorticism.

It is to be expected that remineralization will take place progressively as the

Fig. 3-A: Case 9. A nine-year-old boy with a 30 per cent third-degree burn and skeletal change consisting of periosteal new-bone formation involving both tibiae and both fibulae and ankylosis of both ankles. (In each of the diagrams the shading indicates the area of third-degree burn, and the stars, the area of skeletal involvement.)

Fig. 3-B: Periosteal new-bone formation is present in both tibiae and both fibulae. Both ankle joints are undergoing degenerative change. The roentgenogram was made six months after the burn.

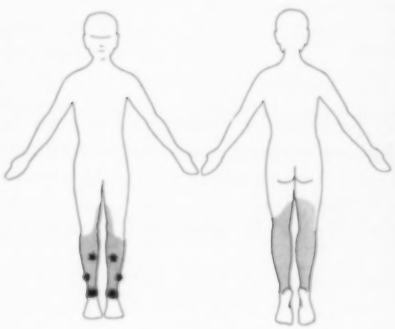

Fig. 3-A

Fig. 3-B

patient is remobilized. We have found in several instances, however, that some degree of atrophy is still evident two or three years after the patient has regained functional independence.

Periosteal New-Bone Formation

This type of reaction is often observed in bone adjacent to a local area of infection or chronic inflammation not associated with burns, and the manifestation as seen in burned extremities is probably no different, since it occurs in those bones which underlie areas of severe burn or in conjunction with a destructive process in an adjacent joint.

It is presumed that the reaction in the badly burned extremity is secondary to the acute hyperaemia or to that accompanying the chronic inflammatory process which persists until there is good skin coverage. Both lower extremities of the patient shown in Figures 3-A and 3-B demonstrate circumferentially open granulating surfaces for four consecutive months. The involved ribs shown in Figure 8-H directly underlie an area which has not yet completely healed. In Case 7 what appears to be irregular ossification of the iliac epiphyses is probably an alteration of the same nature.

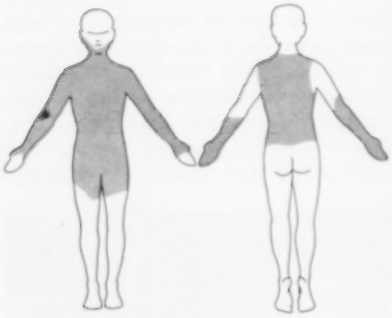

FIG. 4-A

FIG. 4-B

Fig. 4-A: Case 16. A nine-year-old boy with a 55 per cent third-degree burn and pericapsular calcification involving the right elbow.

Fig. 4-B: Discrete, small calcific densities are seen along the medial humeral condyle and in the adjacent capsular ligaments, two months after the burn.

The time of detection of this change has varied from the fourth to the ninth month after the burn. In no instance has there been noted continued proliferation after skin coverage.

Alterations Involving Pericapsular Structures

Pericapsular Calcification

Irregular calcification in the intimately periarticular structures has been an independent finding in seven instances and has accompanied more dramatic change in others. The calcific densities are in the plane of ligaments or overlying tendons and have no attachment to bone. They have been observed in both children and adults. The onset, in the three cases in which it could be established, was in the second and third months after the burn.

In all but one case there has been adjacent or overlying burn. In Case 16 (Figs. 4-A and 4-B) there was symmetrical involvement as far as the burn was concerned, but calcification was limited to the right side. The precipitating causative factor

thus may well be superimposed trauma with punctate hemorrhage and local tissue damage, although these phenomena have not been observed.

This type of spotty calcification may herald more serious joint impairment, it may disappear, or it may remain unchanged about a freely movable joint.

Osteophytes

The most frequently observed skeletal alteration among adult, burned patients is that of the formation of osteophytes. The plaquelike, bone outgrowths which are seen most often at the elbow are called osteophytes because they occur along the articular margins of the olecranon or coronoid process and have the appearance of the osteophytes of osteo-arthritis.

These excrescences limit joint motion and are thus occasionally surgically removed. With surgical exploration and excision it has been determined that they lie in the plane of ligaments, and with histological examination it has been shown that they represent simple new-bone formation in a fibrous connective-tissue stroma.

Osteophytes have been separated from pericapsular calcifications on the basis of appearance alone, the osteophytes being a more disciplined growth that is con-

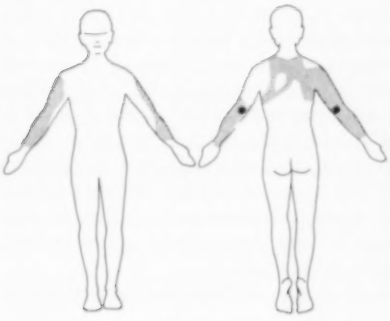

FIG. 5-A

FIG. 5-B

Fig. 5-A: Case 6. A forty-year-old woman with a 17 per cent third-degree burn and osteophyte formation at both elbows.

Fig. 5-B: Three years after the acute burn there are osteophyte-like plaques at the joint margins of the olecranon and coronoid process in the right elbow and complete extra-articular arthrodesis of the left elbow.

sistently attached to bone. The factors provoking the occurrence of the two changes are nevertheless probably much the same. The precipitating cause in the case of the osteophytes could be, for instance, superimposed minor trauma in the form of stress on atrophic ligaments at the site of bone attachment. In every case showing osteophyte formation, there has been an adjacent burn.

These growths may progress to complete bridging of the joint or they may gradually decrease in size. Figure 5-B shows typical osteophytes on the right and ankylosis on the left.

The time of onset has not been determined. Change in all of our cases has been detected after the osteophytic growth has become well established.

Heterotopic Para-articular Ossification

True para-articular osseous bridging has been observed at the hip, elbow, or shoulder in four of our patients, three of whom are children (Figs. 6-C, 6-D, 6-E, 8-B, 8-C, 8-D, and 8-E). These new-bone formations, in the one case explored surgically, have been found to lie deep to and in the plane of surrounding muscle without evidence of actual replacement of muscle substance. The bone bridges in every instance have connection to parent bone at one or both extremes. They have been

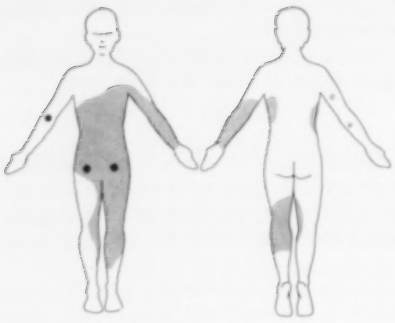

FIG. 6-A

Case 15. A six-year-old boy with a 40 per cent third-degree burn and ectopic-bone formation of both hips and the right elbow.

observed to arise from one bone and extend gradually toward the other, with which they may or may not become continuous. They may cease to grow and remain as simple exostoses, gradually decreasing in size.

The time of onset in Case 15 (Fig. 6) was in the third month after the burn. In other cases, joint motion was already impeded by the new bone before detection, thus the time of onset is not known.

The proximity of the involved joint to the area of burn is not consistently a factor in the production of heterotopic bone. The unburned left elbow shown in Figure 8-C shows extra-articular fusion, whereas the right elbow of the same patient shows marked soft-tissue contracture from an adjacent third-degree burn, but no skeletal change. The situation is similar in regard to the elbows in Case 15 (Figs. 6-D and 6-E).

The pathogenesis of the heterotopic bone may easily be the same as that of the other pericapsular changes. There is evidence, especially in Case 15 (Figs. 6-B and 6-C) where it was recognized, that superimposed trauma is the precipitative factor. It is assumed that, with immobility and progressive bone atrophy, there is a continuing release of bone salts; degeneration of muscle, tendon, and other paracapsular soft tissue is inevitable; and humoral and physiological alterations which follow the acute burn affect alike all soft-tissue structures whether or not they directly underlie the burned area. The severely burned patient thus is constitutionally susceptible to trauma, and it may be that the repeated minor trauma of routine handling or dressing change is a sufficient incitant.

Johnson, in his reporting of this type of change in four patients, three of

FIG. 6-B

FIG. 6-C

Fig. 6-B: This roentgenogram was made two months after an acute burn and at the time of spontaneous dislocation of the right hip. A previous roentgenogram showed no evidence of ectopic bone.

Fig. 6-C: Ten months after injury heterotopic bone, which first appeared one month after the dislocation, has matured. It is attached proximally to the pelvis, and the most anterior spicule lies in the plane of the iliopsoas muscle and appears to articulate distally at the lesser trochanter. There was a minimal change of a similar nature involving the left hip.

FIG. 6-D

FIG. 6-E

Fig. 6-D: This roentgenogram of the right, unburned elbow was made three months after injury and shows heterotopic bone arising apparently from the area of biceps insertion.

Fig. 6-E: In the seventh month after burn the mature structure of the new bone is evident. From this stage it regressed in size as the general condition of the patient improved.

whom had received third-degree burns, also noted the occurrence of heterotopic bone in unburned areas and implicated the trauma of routine care. He referred to the change as atypical myositis ossificans, and the two processes may indeed be similar in every respect. Heterotopic-bone formation has been noted in patients with poliomyelitis and with paraplegia, and there is nothing specific to distinguish it from that seen in burns. The identification of the process with myositis ossificans, however, does not make the actual pathogenesis less obscure, and, as with myositis ossificans, there must be factors other than trauma because among severely burned patients receiving the same type of care there are many in whom heterotopic bone does not develop.

It is not within the scope of this preliminary report to discuss the various theories of ectopic-bone formation, nor to speculate further as to the cause in burned patients. It is our hope, however, that continued study of these individuals will to some degree clarify the problem.

Alterations Involving the Joint

Articular Destruction and Ankylosis

Progressive destruction of one or more joints with eventual fibrous or bony ankylosis has occurred in seven patients, all of them children. The sequence of this articular change is illustrated in Case 14 (Figs. 7-B, 7-C, and 7-D) and in Case 3 (Figs. 8-F and 8-G).

The earliest clinical evidence thus far observed is fusiform periarticular swelling and local heat, and the affected joints are painful on palpation and manipulation. This finding is not in itself a particularly unusual one in a badly burned child. The time of onset may be one or several months after the acute burn.

The early roentgenographic manifestations are those of capsular swelling, spotty pericapsular calcification, and subchondral irregularity (Fig. 7-B). As previously mentioned, the calcifications may or may not portend joint destruction.

The advancing process, as observed on roentgenograms, shows soft-tissue and intracapsular clouding, progressive destruction of articular surfaces, adjacent reactive periosteal new-bone formation, and, ultimately, ankylosis (Figs. 7-C, 7-D, 8-F, and 8-G). Complete articular destruction may occur within a few weeks, and ankylosis within a few months.

The joints most frequently involved are the elbow, hip, and ankle. Talocalcaneal ankylosis has occurred in two instances in which there is adjacent ankle fusion, and fibrous ankylosis has been observed in one knee (Table I, Case 2).

The proximity of the joint to the burned area is not a constant factor in the genesis of intra-articular change. In Case 3 (Figs. 8-D and 8-E), the involved left hip underlies an area of a third-degree burn but the involved right ankle (Figs. 8-F and 8-G) is far distal to the nearest third-degree involvement on the thigh. In Case 14 (Fig. 7-D), ankylosis has occurred in the elbow on the slightly burned right side, whereas there is normal elbow function on the burned left side.

It is our opinion that in its behavior this process more closely resembles septic arthritis than any other recognized entity. The possibility that the destruction is due to bacterial invasion is supported by the knowledge that all severely burned patients have a recurring transient bacteraemia or septicaemia of varying severity as long as there are open granulating areas. Artz and Reiss made a rather definite assertion that the joint destruction in burned patients is due to infection. Although we, too, incline to this opinion, we lack confirming evidence which may in time be gained from autopsy and surgical specimens or from aspiration of joints, and there is clinically no positive correlation between the amount and degree of sepsis and involvement of joints. The examination of two ankles in surgically removed lower extremities with almost total, deep, third-degree burn disclosed pannus

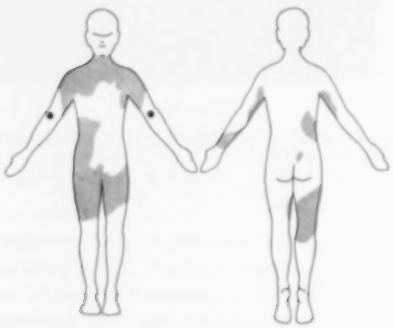

FIG. 7-A

Case 14. A seven-year-old girl with a 40 per cent third-degree burn and a skeletal change of pericapsular calcification of the left elbow and ankylosis of the right elbow.

FIG. 7-B

FIG. 7-C

FIG. 7-D

Fig. 7-B: A roentgenogram of the slightly burned, right elbow, made three months after injury, showing small calcific densities apparently capsular in location and periosteal new-bone formation along the anterior surface of the humerus adjacent to the joint. The fusiform soft-tissue swelling is evident.

Fig. 7-C: The periosteal new-bone formation is pronounced, and there is marked articular destruction four months after injury.

Fig. 7-D: This roentgenogram, fifteen months after injury, shows complete clinical ankylosis of the right elbow.

formation and fibrinous adhesions without suppuration. This finding suggests that a degenerative process could be initiated without bacterial invasion.

SURGICAL EXPERIENCE

If osteophytes or heterotopic bone severely limits joint motion, excision of the offending growth is indicated. We have removed osteophytes from the olecranon and coronoid process of the ulnae in Cases 6, 7, and 12 (Table I). Heterotopic bone of such extent as to cause extra-articular arthrodesis has been removed from the right hip and the left elbow of Case 3.

The removal of osteophytes resulted, in all three cases, in a restoration of functional range of motion. In each of the cases it was necessary after excision of the bone block to manipulate the joint forcibly in order to break down soft-tissue adhesions. Supervised active exercise was begun in the first week after operation with emphasis on strengthening. Range of motion tended to increase gradually. There has been no recurrence of the osteophytes.

In Case 3, however, heterotopic bone has recurred at the hip, but without complete bridging so that there is at least a limited range of motion in all planes, eighteen months after surgery. Removal of the wedge of bone at the elbow has enabled the patient to put her left hand to her face, and there has been no recurrence at this site.

In Case 1, an arthroplasty of the left hip, utilizing a Judet prosthesis, has been only temporarily effective in restoring motion. Simple corrective osteotomies of the left femur in Case 3 and of the right knee in Case 2 have been most helpful.

It is our policy to wait until the burn scar over the skeletal area to be approached is pale, soft, and pliable before planning a surgical procedure. Ideally, elective surgical procedures should be postponed until there is no granulating surface or scar tissue in any location regardless of the condi-

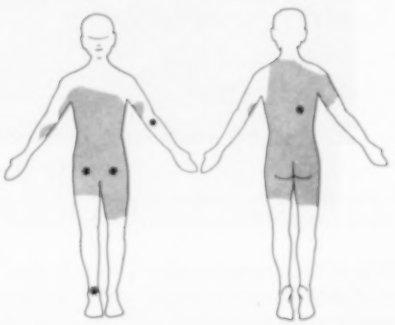

FIG. 8-A

Case 3. A six-year-old girl with a 50 per cent third-degree burn. The skeletal change consists of periosteal new bone of the ribs, ectopic bone of the right hip and left elbow, and ankylosis of the right ankle and left hip.

FIG. 8-B

FIG. 8-C

Fig. 8-B: Nine months after the acute burn there is heterotopic new bone bridging the unburned left elbow joint in the plane of the brachialis muscle.

Fig. 8-C: At seventeen months there is complete extra-articular bony ankylosis.

FIG. 8-D

Fig. 8-D: Nine months after the acute burn there is intra-articular destruction of the left hip and progressive extra-articular osseous bridging of the right hip.

tion of the operative site. It is our observation that as long as there is activity of scar or granulation tissue any focus of ectopic bone, regardless of its location, will continue to proliferate and thus the chances of recurrence of such ectopic bone after its removal are probably enhanced. Under no circumstance should an incision be made through a scar that is red or indurated. These two qualities indicate that the scar is active or that there is still an inflammatory response in the area. Closure of an incision through an indurated scar is difficult; the wound edges tend to necrose, and the scar is made worse.

In children, if heterotopic bone merely limits motion but does not cause ankylosis of the joint it should not be excised. As the patient's general condition improves and the scar tissue softens, ectopic growths tend to decrease in size, and joint motion is gradually restored.

GENERAL OBSERVATIONS

Direct Involvement

If an area of bone is denuded by burn there results tangential sequestration of the avascular portion (Case 20). This necrotic bone will remain exposed until by means of drilling or tangential sequestrectomy granulation tissue is encouraged to grow and provide a bed for a skin graft. Moncrief has adequately covered this problem in his paper dealing with complications of burns. Glover called attention to bone necrosis resulting from direct involvement of bone or periosteum by burn. We have not yet observed the type of segmental necrosis which occurred in one of his cases.

Direct involvement of joints usually results in septic arthritis, articular destruction, and ankylosis. The joints most frequently involved are those of the wrist and hand. In one patient (Table I, Case 10), the wrist was involved directly.

FIG. 8-E

The mature alterations of intra-articular ankylosis on the left and extra-articular ankylosis on the right are shown as they appeared six years after injury.

FIG. 8-F

FIG. 8-G

Fig. 8-F: Seven months after the burn there is articular destruction involving the talotibial and talocalcaneal joints, intracapsular cloudiness, and adjacent reactive periosteal new-bone formation in the unburned right ankle.

Fig. 8-G: The talotibial and talocalcaneal bony ankylosis as it appeared six years after the burn.

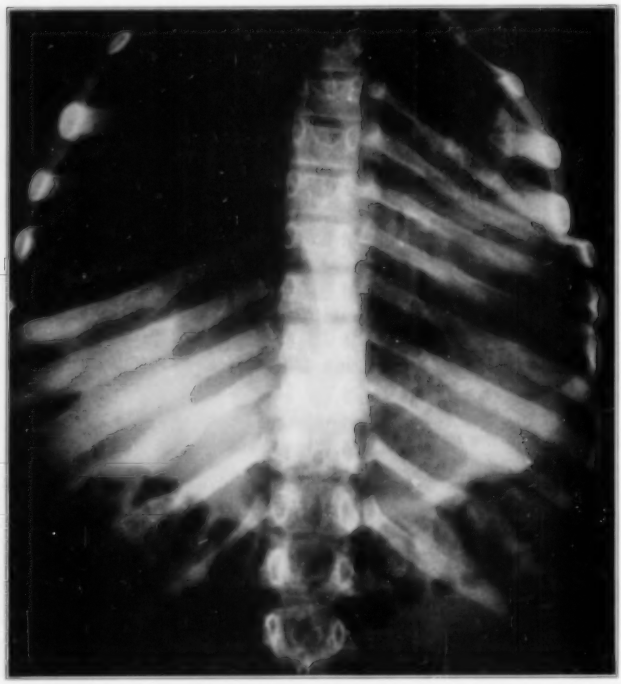

FIG. 8-H

The widened and fused ribs on the right are the result of reactive periosteal new-bone formation. The roentgenogram was made six years after the burn, which involved the skin overlying this area.

Distribution and Type of Alteration

Skeletal changes after a burn have been observed to occur in all major joints of the extremities. The interphalangeal, metacarpophalangeal, and metatarsophalangeal joints have not shown these changes in the cases reviewed, although they have been involved by the burn directly. Hips and elbows are frequently the sites of involvement, but shoulders, wrists, and knees are rarely involved in spite of their frequent inclusion in the burned area. Among the cases cited by Kolář and Vrabec, calcification about the knee was a rather common finding.

It has been a consistent observation that joints with major para-articular changes do not show intra-articular changes and that the converse is true. A patient may demonstrate only one major type of alteration, as in Case 15 (Figs. 6-B through 6-E), or he may show both intra-articular and para-articular changes in different joints of the same extremity as in Case 3 (Figs. 8-B through 8-G).

Age and Severity

Skeletal alteration after burns has been observed to occur more often and to a greater degree in children than in adults. The most severe response so far observed in an adult is that of heterotopic ossification in the shoulder of a woman twice burned (Case 13). In no instance has intra-articular destruction or ankylosis been observed in an adult.

This discrepancy in the incidence of skeletal change between children and adults who have been burned cannot be explained on the basis of differences either in the frequency of burn or in survival rate. For want of a better explanation, we ascribe this difference to the exuberant tissue response characteristic of youth. However, since the phosphate concentration in the serum of children is twice that

of adults, it appears reasonable to presume that the differences noted in the incidence of calcium deposition and heterotopic-bone formation are in some way related to this.

Predisposition to Change

We have noted that of the 950 patients with burns of all severity treated in John Sealy Hospital from January 1950 through August 1958, only twenty have demonstrated skeletal alteration other than osteoporosis. We are quite sure that a number of cases have been overlooked because the skeletal involvement did not sufficiently limit function to warrant investigation, but it is still apparent that major skeletal changes do not develop in most burned patients. Thus, as with similar changes in non-burned patients, there is likely to be an individual or hereditary predisposition.

Growth Stimulation

We are occasionally aware of growth spurts in children after severe burns, and there are adult patients who report that they required larger shoes after a burn than they did before. One patient, who was twenty-four years of age at the time of the burn, gained approximately one and one-half inches in height after the burn and reported an increase of one full shoe size.

It is possible that a severe burn may stimulate skeletal growth through stasis or passive hyperaemia or a chronic inflammatory process. To bring random reports and clinical impression to scientific soundness, however, would require routine teleoroentgenographic study, a mechanically unfeasible undertaking in the severely burned patient. We therefore simply wish to record the observation.

SUMMARY

The skeletal alterations which occur in association with thermal burns have been arbitrarily classified according to their roentgenographic appearance as: osteoporosis, periosteal new-bone formation, pericapsular calcification, osteophyte formation, heterotopic para-articular ossification, joint destruction, and ankylosis.

Possible explanations for each of the skeletal changes are offered, but the true pathogenesis is still obscure. Superimposed trauma may be a precipitating factor in instances of heterotopic calcification and ossification and sepsis may be a factor in intra-articular change.

Surgical excision of ectopic osseous or calcified tissue is a feasible procedure but should not be undertaken until the patient's general condition is good and all scar tissue is mature and soft.

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Effects of Rotatory and Valgus Malpositions on Blood Supply to the Femoral Head

Observations at Arthroplasty *

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In subcapital fractures of the hip, circulation to the proximal fragment may be impaired or completely interrupted by varying degrees of rotation of the femoral head. An extreme valgus position of the capital fragment may also embarrass this blood supply. These observations were first suggested to me thirteen years ago during the performance of an osteosynthesis reconstruction operation on the hip. The patient was a woman, fifty-three years old, who had an established non-union of a high subcapital fracture of the right hip. Since the femoral neck was well preserved and the head was apparently viable, it was decided to carry out a Brackett procedure. When the capsule was opened and the non-union cleaned out, the freshly denuded surface of the capital fragment bled freely. I was impressed by the fact that the bleeding took place from all parts of the femoral head. The blood dripped actively at a measured rate of seventy to eighty drops per minute.

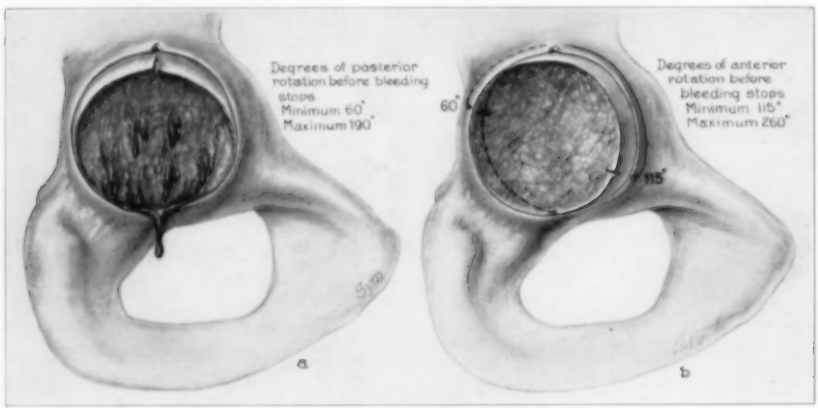

Fig. 1

Symmetrical head fragment *in situ*. *A:* Position of maximum active bleeding. Notches made in superior portion of the head and adjacent acetabular rim are points of reference for rotational studies. *B:* Bleeding stopped when head was rotated anteriorly to 115 degrees and beyond. Bleeding also stopped at and beyond 60 degrees of posterior rotation.

In testing its mobility, the symmetrical head fragment was turned first clockwise then counterclockwise in the plane of the acetabular margin. As the turning was increased in either direction, the bleeding gradually slowed and finally stopped. On returning the head fragment to its original position, the dripping of blood recurred at approximately the initial rate. The head was then tilted into various other positions. Extreme valgus was the only position which shut off the blood supply completely.

In this patient there were no remaining retinacular attachments to the head fragment. Therefore, its only blood supply entered through the ligamentum

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teres. It seemed significant that the blood supply through the ligamentum teres could be interrupted at will by placing the head fragment in rotational and valgus malpositions. Furthermore, if these malpositions of the head fragment could embarrass, if not obstruct, the only blood supply available to the femoral head, could not such positions be contributing factors in many cases of non-union, delayed union, early or delayed aseptic necrosis, and degenerative arthritis?

Stimulated by this experience, I looked on each subsequent open operation on the hip joint as an opportunity to test these initial observations.

OBSERVATIONS AT OPERATION

During the past thirteen years, twenty-four patients had reconstruction operations of the hip which afforded opportunities to look for bleeding from the femoral-head fragments. While some heads had residual shreds of retinacular attachments, none had patent retinacular circulation. Before testing the capital fragments for bleeding, any remaining retinacular attachments were sacrificed. This left each head fragment freely movable in the acetabulum except for its attachment to the ligamentum teres. The observations made at operation can be divided into three categories: effects of rotatory movement, effects of movement other than rotational, and bleeding from the cut ligamentum teres.

Effects of Rotatory Movement

In a sense, all movements of a ball-and-socket joint are rotational. However, as used in this paper, the term *rotatory movement* refers only to movement of the femoral-head fragment in the plane of the acetabular margin around an axis passing through the head at or near the fovea centralis.

Of the twenty-four hip operations, six were Brackett reconstructions, one was a Whitman reconstruction, three were high femoral-neck osteotomies, and fourteen were insertion of a prosthesis (three primary). In each instance an attempt was made to establish:

1. The presence of active bleeding from the capital fragment;
2. The rate of such bleeding (drops per minute);
3. The size of the surface area of the fragment contributing to the bleeding;
4. The degree of rotation of the head, in the plane of the acetabular margin, necessary to interrupt active bleeding.

The technique of examining the capital fragment for evidence of bleeding consisted in removing bone bit by bit with a rongeur. In some instances it was necessary to remove only the fibrocartilaginous covering of non-union. In others, from one-fourth to one-half inch or more of the cancellous bone was removed from the head before an active blood supply was tapped.

There was wide variation in the amount of spontaneous bleeding from the capital fragments. This variation was attributed to the degree of patency of the foveal vessels or of the intra-osseous vascular channels or of both.

Twenty-four head fragments were examined *in situ*. Of these: Four fragments were completely avascular throughout. Twelve fragments exhibited areas of fresh blood that dripped only occasionally. After a few minutes the dripping ceased and did not start again until the surface of the fragment had been cleaned of clotted blood or additional bone had been removed. Although certainly indicative of viability, such bleeding was considered too inactive for rotational studies. The remaining eight head fragments bled actively enough for testing. Six bled from all surfaces and two bled only from the antero-inferior half. The amount of bleeding varied from a minimum of twelve drops per minute to a maximum of eighty drops per minute.

The position of each head fragment in the acetabulum at which maximum bleeding occurred was selected as the starting point for studies on the effects of rotatory movement. In this position a notch was made in the superior acetabular margin. Another notch was made in the adjacent superior margin of the head

fragment. Thus, the second notch was directly below the first, an arrangement which ensured return to the original position after rotation for measured distances (Fig. 1, *A*).

Rotation was measured in the plane of the acetabular rim and was referred to as either anterior or posterior. Anterior rotation was designated as the distance traveled by the notch in the superior margin of the head fragment along the anterior acetabular margin in the direction of the symphysis pubis. Posterior rotation was designated as the distance the notch in the superior head fragment moved along the posterior acetabular margin in the direction of the posterior ischium. Rotation was expressed in degrees of a circle.

Each head fragment was slowly rotated anteriorly until active bleeding from its surface stopped. The number of degrees the notch on the head fragment had moved along the anterior acetabular rim was recorded. Then the head fragment was returned to its original position. When active bleeding recurred, the rotation procedure was repeated, but in a posterior direction.

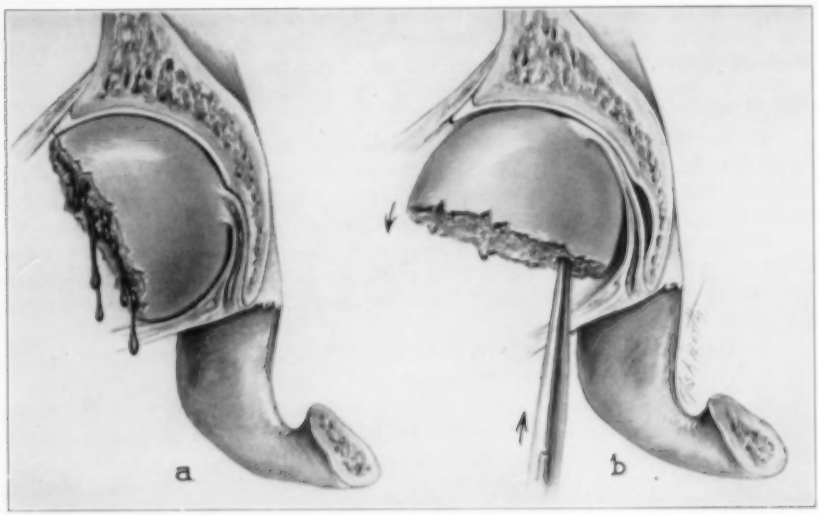

FIG. 2

Coronal section of acetabulum containing symmetrical head fragment. *A:* Actively bleeding head. Ligamentum teres and open foveal vessels in anatomical position in fossa. No rotation of head and no twisting or stretching of ligament. Situation comparable to sitting or quadruped position shown in Fig. 3. *B:* Bleeding stopped by tilting head fragment in the extreme valgus position. The ligamentum teres and its vessels have left the fossa superiorly, are overstretched, and are compressed between the lunate surface of the acetabulum and femoral head.

As one might suspect, the number of degrees of rotation necessary to shut off the blood supply from the ligamentum teres varied greatly in different head fragments. The minimum excursion anteriorly was 115 degrees; the maximum was 260 degrees—almost three-quarters of a circle. Rotation posteriorly stopped active bleeding at a minimum of 60 degrees and a maximum of 190 degrees (Fig. 1, *B*). These variations were ascribed to observed differences in the length and thickness of the ligamentum teres as well as in the depth of the acetabular fossa, as will be explained later.

In two instances the head fragments were not large enough for reconstruction, although adequate for testing the degrees of rotation necessary to shut off active bleeding. One of these could be delivered out of the acetabulum without interrupting the flow of blood. This was first tilted into varus position and then dislocated out of the acetabulum antero-inferiorly over the acetabular notch. By this

route minimum tension was placed on the ligamentum teres and the foveal vessels. Once delivered out of the acetabulum, it was possible to see the ligamentum teres throughout its entire length. This situation offered the only opportunity in the series to rotate the dislocated head fragment and simultaneously watch the twist increasing in the ligamentum teres to the point where bleeding stopped.

Effects of Movements Other than Rotational

Each of the eight head fragments was also tested to see if its bleeding would be affected by movements other than rotational, namely, anteversion, retroversion, varus, and valgus. The results were as follows:

Extremes of anterior and posterior displacement of the head, comparable to anteversion and retroversion, slowed and finally stopped bleeding in six of the eight heads. In the other two heads bleeding continued slowly.

With the head in the position of maximum active bleeding, the superior margin of the head fragment was depressed in the acetabulum, thereby tilting the head into extreme varus position. In this position the bleeding was slowed in several heads, but remained active in all.

Tilting the fragment into slight valgus position had no effect on bleeding. Moderate degrees of valgus position caused partial interruption of bleeding. Tilting the fragment into extreme valgus deformity abruptly and completely stopped bleeding in all eight heads (Fig. 2). On return to the original position, active bleeding resumed in seven of the eight heads. Bleeding ceased in one and would not start again despite the removal of bone and cleaning of the bleeding surface. In this instance the ligamentum teres, presumably, had been structurally damaged and the foveal blood supply permanently interrupted by the testing maneuver. Fortunately, this occurred in a patient for whom the primary insertion of a prosthesis had been planned.

Bleeding from Cut Ligamentum Teres

During the period reviewed, thirty-two additional reconstruction operations of the hip were done (seventeen arthrodeses and fifteen cup arthroplasties). These, together with twelve operations from the group of twenty-four (eleven insertions of a prosthesis and one Whitman reconstruction), made a total of forty-four operations in which the femoral head was removed.

In each of these forty-four operations an effort was made to determine the presence or absence of gross bleeding from the stump of the ligamentum teres remaining in the acetabular fossa. Some degree of gross bleeding was demonstrated in thirty-three instances (70 per cent). Nineteen stumps bled actively enough to require cauterization. Four bled spontaneously but did not require cauterization. In the other ten stumps there was no spontaneous bleeding immediately following dislocation of the femoral head; but, in all ten bleeding could be initiated either by manipulating the crushed end of the stump or by dividing the ligament closer to the acetabular notch.

ANATOMICAL STUDIES OF THE LIGAMENTUM TERES

To clarify the notations made at operation on living subjects, related anatomical studies were also carried out. Some were made on the hip joint *in situ* at autopsy. Others were done on resected hip joints. Before presenting the details of these studies, it might be well to review, briefly, anatomical considerations pertaining to the acetabular fossa as well as to the ligamentum teres.

The fossa, located in the antero-inferior portion of the acetabulum, is roughly square. It makes up approximately one-third to one-half of the entire acetabulum. Its floor is depressed below the surrounding lunate cartilaginous surface. This lunate surface is in contact with the cartilaginous surface of the femoral head and creates the anatomical space of the fossa. The depth of the fossa varies, but easily accommodates its ligamentum teres.

FIG. 3

FIG. 4

Fig. 3: Intact hip in approximately 90 degrees of flexion (sitting or quadruped position). Ligament lies midway in fossa, is flat throughout, and is directed superiorly. Each margin seems equally stretched (with no overstretching). Origin and insertion are almost parallel. It was in this position that maximum bleeding occurred from the head fragments (Fig. 1, *A*).

Fig. 4: Maximum acute flexion of intact hip. No rotation. Anterior margin of ligament stretched; posterior margin relaxed. Insertion line is rotated 60 degrees posteriorly to line of origin. Ligament occupies mid-portion of fossa directed posterosuperiorly.

The ligamentum teres is a straplike structure draped over the convex cartilaginous surface of the femoral head. It is a composite anatomical unit made up of the ligament itself, the foveal vessels, areolar tissue, and some fat—all wrapped in a tube of synovial membrane extending from the fovea to the acetabular notch. Hereafter when reference is made to alterations of the ligament, it will be necessary to visualize that they involve this synovial tube and all its contents.

Studies on Hip Joints in Situ

Three hip joints were explored in fresh cadavera through the intra-articular approach. In each instance a subcapital fracture was created by osteotomy through the medial portion of the femoral neck, just proximal to the retinacular attachments. Each symmetrical head fragment was then dislocated antero-inferiorly out of its acetabulum, leaving the ligamentum teres intact. The three ligaments varied in length and thickness, one being short and thick, the other two, longer and thinner.

Each dislocated head with its ligament attached was tested in the following manner: The head was rotated around its foveal axis—first clockwise, then counterclockwise. As the degree of rotation increased in either direction there was a correspondingly greater degree of twisting in the ligamentum teres and, finally, gross distortion of its original contour. In other words, the ligamentum teres became progressively shorter, thicker, and more irregular until it resembled a tightly wadded, lumpy mass which resisted further rotation of the head.

The degree of rotation required to reach the maximum point of twisting, without tearing the ligament, varied according to the structure of the ligamentum teres. Thus, the two longer and thinner ligaments allowed a full half-circle more of rotation of the head than did the shorter and thicker ligament.

After untwisting its ligamentum teres, each head was replaced in its original position in the acetabulum. The replaced head was then rotated around its foveal axis and in the plane of the acetabular margin. When the head was held snugly in the acetabulum the maximum rotation was only one-half to two-thirds as great as that which had been achieved with the dislocated head. When the head was held less snugly in the acetabulum further degrees of rotation were possible, but eventually resulted in extrusion of the head from the acetabulum.

These observations were attributed to the progressive compression of the ligamentum teres. This compression was brought about by the growing disproportion between the fixed depth of the acetabular fossa and the changing shape of the wadded portion of the ligament.

It was soon apparent that the functional anatomy of the ligamentum teres could not be studied adequately through the intra-articular approach alone. Therefore hip joints were removed from fresh cadavera and specially prepared for further studies.

Studies on Resected Hip Joints

Each specimen consisted of a hip joint removed intact with its innominate bone and proximal part of the femoral shaft. In preparing the specimen, all soft tissue was removed from the innominate bone and femur, with the exception of the capsule which was left intact. The hip joint was exposed from the pelvic side by removing the entire floor of the acetabular fossa, care being taken that none of the acetabular cartilage was removed. The synovial membrane was developed as a flap, which was turned antero-inferiorly and medially away from the margins of the fossa toward the acetabular notch. When the synovial flap was reflected, a window was opened which allowed a full view of the ligamentum teres and that portion of the femoral head which normally comes into contact with the acetabular fossa.

To preserve the specimens in a pliable state for the studies, they were wrapped in cloths saturated with normal saline solution and refrigerated.

Specimens thus prepared permitted direct observation of the ligamentum teres

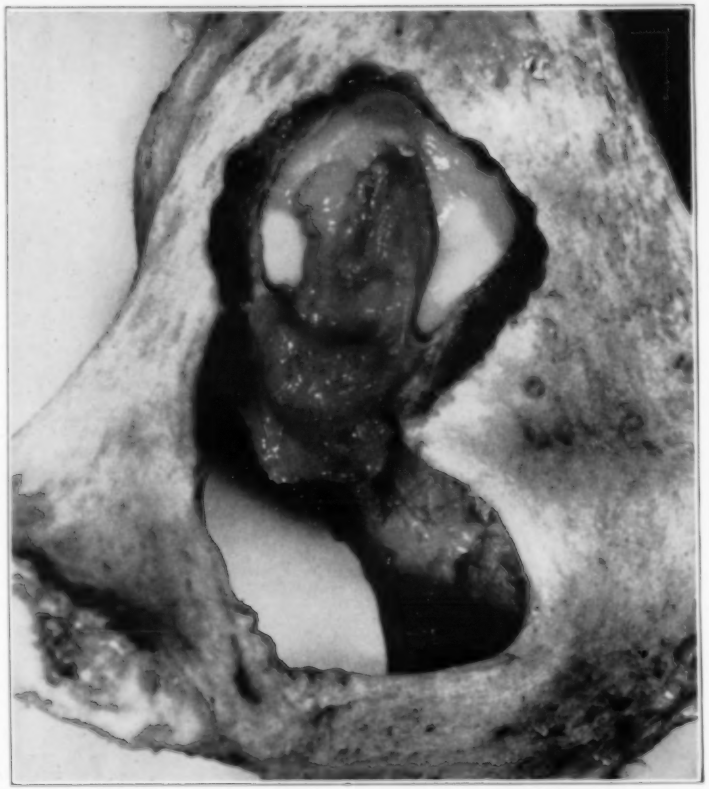

FIG. 5

FIG. 6

Fig. 5: Standing position of intact hip. No rotation. Ligament occupies mid-position of fossa and is directed superiorly. Posterior margin is moderately stretched; anterior margin is relaxed and redundant. Insertion is rotated anteriorly about 70 degrees from line of origin.

Fig. 6: Intact hip is fully extended and externally rotated. Ligament is placed antero-inferiorly in fossa. Insertion in head is rotated anteriorly 85

FIG. 7

Moderately acute flexion and internal rotation of intact hip. Ligament occupies inferior half of fossa and is fully stretched. Insertion is rotated 30 degrees posteriorly from line of origin.

during studies made on both normal and abnormal ranges of movement of the femoral head.

Normal Ranges of Movement

In the specimens used for these studies, continuity of the femur was not disturbed and the capsule remained intact and normally pliable. The excursions of the head and ligamentum teres in these specimens were therefore kept within physiological limits. The relationship of the teres to the fossa in its functional anatomy is shown in Figures 3 through 7.

In flexion and extension of the intact hip, the head rotated more nearly in the plane of the acetabular rim. At approximately 90 degrees of flexion of the hip the ligament was flattened and had little or no twist. In this position (sitting or quadruped) the ligamentum teres had its most anatomical appearance (Fig. 3).

In acute flexion there was approximately 60 degrees of posterior rotation of the femoral head. As a result, the ligamentum teres was slightly twisted at its insertion (Fig. 4).

In the standing position or in supine recumbency, the head rotated anteriorly approximately 70 degrees from the quadruped position. In so doing, the ligamentum teres was moderately twisted (Fig. 5).

In hyperextension the head rotated further anteriorly, approximately 85 to

90 degrees. Consequently, there was more twisting of the ligamentum teres (Fig. 6).

In external rotation (moderate anteversion) the ligamentum teres and fovea moved forward and occupied positions more nearly related to the anterior margins of the fossa (Fig. 6).

Internal rotation of the normal hip (moderate retroversion) placed the foveal end of the ligament in close contact with the posterior margins of the fossa (Fig. 7).

Adduction of the femur tipped the head into a slight degree of valgus position. This caused slight stretching of the ligamentum teres as the fovea moved superiorly in, but not out of, the fossa.

These observations indicate that in normal ranges of hip movement the fovea capitis femoris, together with the insertion of the ligamentum teres, may occupy any part of the roughly square acetabular fossa. Furthermore, it is impossible for the fovea or any significant part of the ligamentum teres to leave the area of the fossa. This remains true as long as the femoral head maintains continuity with the neck which, in turn, is controlled by an intact hip-joint capsule.

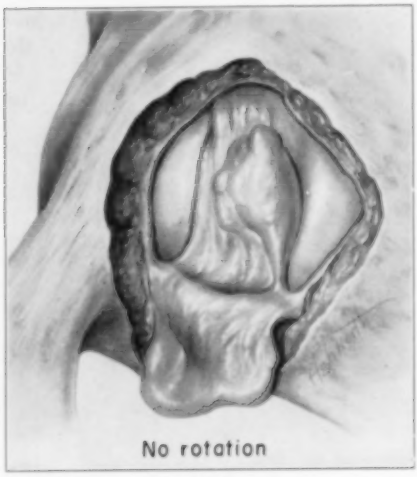

FIG. 8

Position of head fragment is comparable to that of sitting or quadruped position of intact hip, as seen in Fig. 3. Ligament is relaxed, without twisting or stretching. This is position of maximal bleeding (Fig. 1, *A*). This was the starting position for rotational studies.

Abnormal Ranges of Movement

One of the hip specimens previously studied was artificially fractured in the subcapital area. This created a symmetrical proximal head fragment. Uninfluenced by bone or soft-tissue attachments to the neck, the head was free to move in abnormal ranges. Studies were confined to the effect of abnormal ranges of rotation and valgus position on the ligamentum teres.

The abnormal degrees of rotatory movements made by the free head fragment were drawn by an artist from direct observation and photographs of the specimen. These show the amount of twisting in the ligamentum teres at each 90-degree increment of rotation anteriorly and posteriorly (Figs. 8 through 10-C).

Rotation was started with the head fragment in the position shown in Figure 8. This is the position in which the ligament is flattened and untwisted. It is also the position which seemed to allow maximum active bleeding from each of the heads observed at arthroplasty. From the starting position, increasing degrees of anterior rotation of the femoral head resulted in the progressive twisting deformity of the ligamentum teres shown in Figures 9-A through 9-D. From the same starting position, rotation of the head fragment posteriorly resulted in increasing degrees of twisting of the ligamentum teres in the opposite direction (Figs. 10-A, 10-B, and 10-C).

As clearly shown in these drawings, increasing degrees of rotation in either direction produced greater amounts of twisting of the ligament. With increasing twisting the ligament became thicker and shorter, and finally doubled on itself. Since the depth of the fossa remained constant, it is easy to see how portions of the ligament, twisted into increasing bulk, became compressed. The progressively greater amount of twisting and the compressing force of the unchanging space of the fossa could easily account for the cessation of foveal blood supply to the head fragment.

Tilting the free head fragment into extreme valgus position caused the fovea to move superiorly out of the acetabular fossa. As a result, the ligamentum teres became taut, and its insertion was compressed between the cartilaginous surface of the head and the lunate cartilage of the acetabulum (Fig. 11). This situation would lead to complete interruption of the foveal blood supply.

DISCUSSION

No other fractures have received as much attention as intracapsular fractures of the hip. This is particularly true of the more medial subcapital fractures. Yet, paradoxically, their management universally yields a high percentage of poor results. There would seem to be two main reasons for this: the mechanical disadvantages peculiar to the bone anatomy of the area and the vulnerability of the blood supply to the femoral head. During the past twenty-five years significant

FIG. 9-A

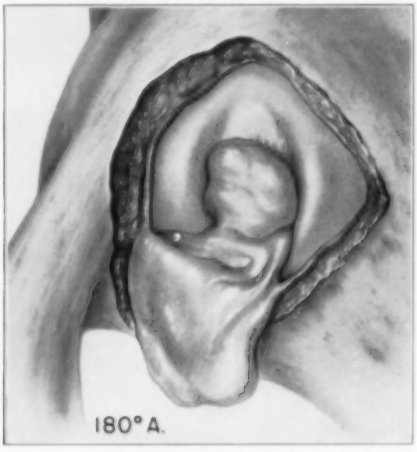

FIG. 9-B

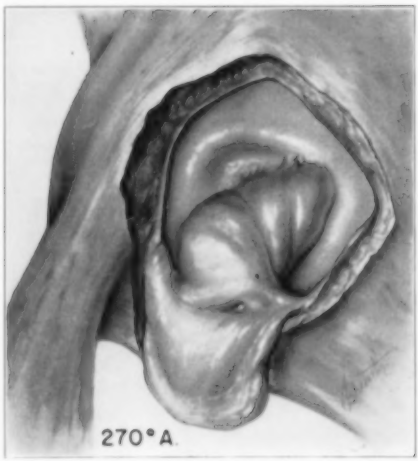

FIG. 9-C

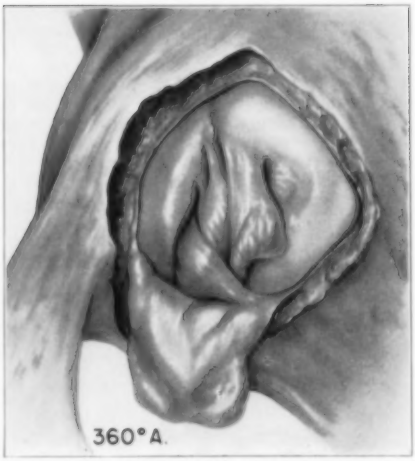

FIG. 9-D

Figs. 9-A through 9-D: Note progressive increase in twisting, shortening, and thickening of ligamentum teres resulting from 90 degrees, 180 degrees, 270 degrees, and 360 degrees of anterior rotation.

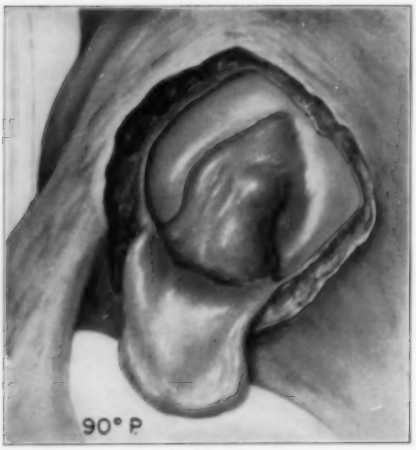

FIG. 10-A

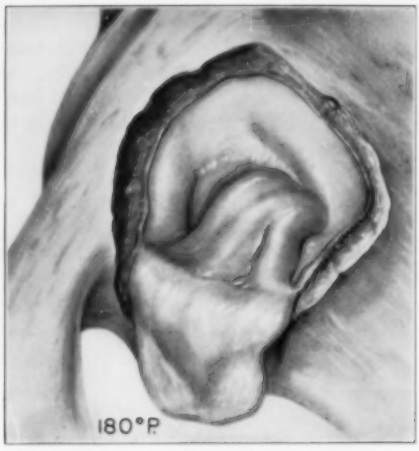

FIG. 10-B

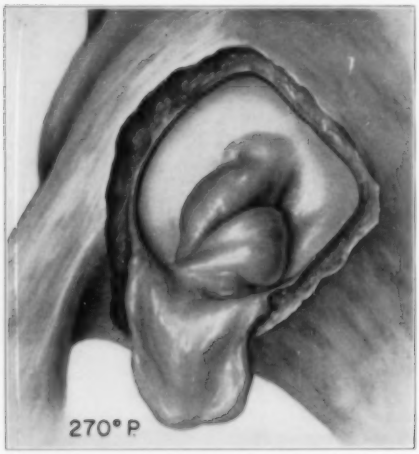

FIG. 10-C

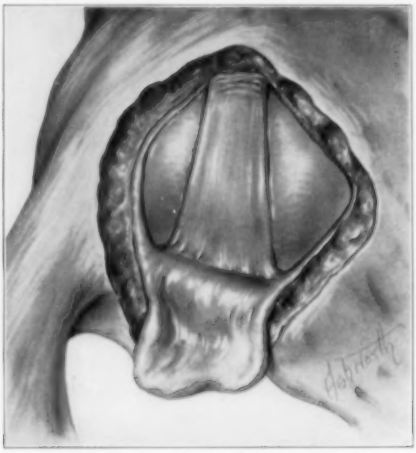

FIG. 11

Figs. 10-A through 10-C: Note progressive increase in twisting, shortening, and thickening of ligament resulting from 90 degrees, 180 degrees, and 270 degrees of posterior rotation.

Fig. 11: Capital fragment has been tilted into an abnormal degree of valgus position. This has drawn the ligament taut over the convex surface of the head. The fovea with its teres insertion has moved superiorly out of the fossa. Ligament is compressed at the superior margin of fossa; its loose synovial covering is thrown into folds.

advances have been made in our knowledge of this fracture. Nevertheless, *the unsolved fracture* is still not a misnomer.

Since the introduction of the three-flanged nail for intra-osseous transfixation of these fractures, treatment has gradually become more uniform. Today most of us would agree that the basic criteria of treatment are:

1. Reduction at the earliest possible moment;

2. Avoidance of further trauma to the fracture site, before and during reduction;

3. Reduction anatomically or in slight valgus position, as determined by anteroposterior and lateral roentgenograms;

4. Maintenance of reduction until there is evidence of bone union and, perhaps, for a longer period.

The degree to which these criteria are followed varies, naturally, with each

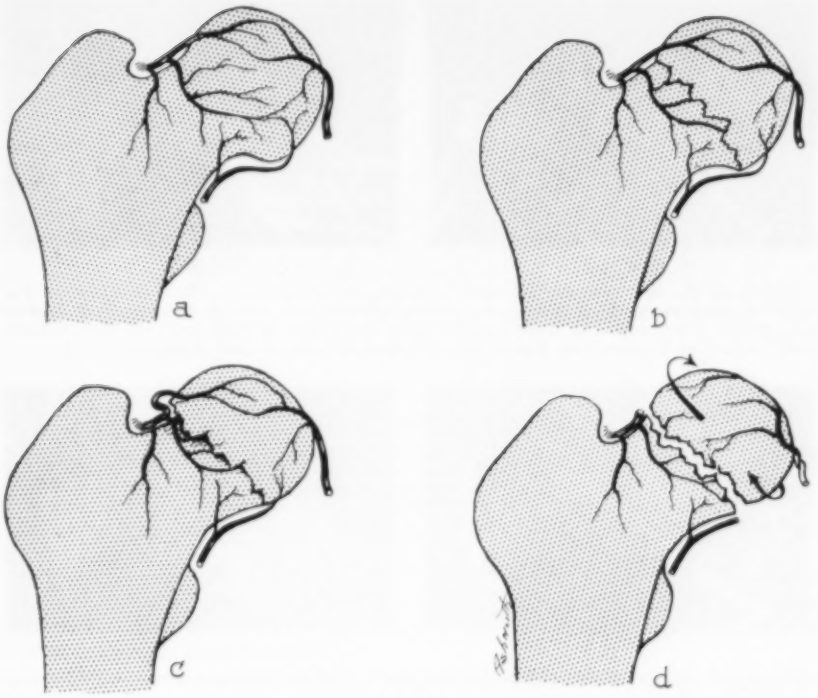

FIG. 12

Schematic representation of blood supply to the femoral head as affected by different types of subcapital fractures. *A:* Intact femoral head with uninterrupted blood supply through the intra-osseous, retinacular, and ligamentum teres vessels. *B:* Lineal subcapital fracture without displacement resulting in interruption of intra-osseus blood supply only. *C:* Subcapital fracture impacted in moderate valgus position without rotation. Retinacular and teres blood supplies intact. *D:* Subcapital fracture with free femoral head in rotatory malposition. Head is avascular because rotation has resulted in tearing of the retinacular vessels and severe twisting of the ligamentum teres.

surgeon. The individual variations are most evident in postoperative management, particularly regarding the time at which weight-bearing is first allowed. Yet even the most meticulous surgeon is too often dissatisfied with his results.

In addition to maintenance of reduction, good results presuppose an adequate blood supply to the fracture fragments. Anything which happens at the fracture

FIG. 13-A

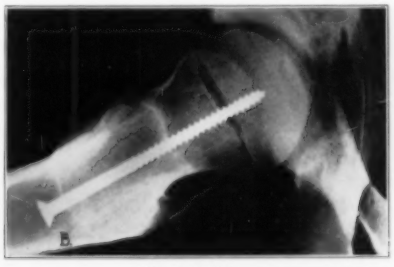

FIG. 13-B

Subcapital fracture. Symmetrical head in 90 degrees of anterior rotatory malposition. Fig. 13-A: Anteroposterior view. Continuity of pattern of stress trabeculae from head to neck across fracture line. Fovea, barely visible, opposite acetabular fossa. Fig. 13-B: Lateral view. Trabecular pattern of head and neck continuous across fracture. Fovea not seen.

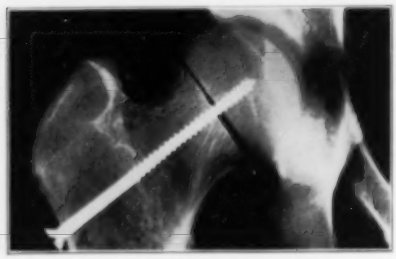

FIG. 14-A

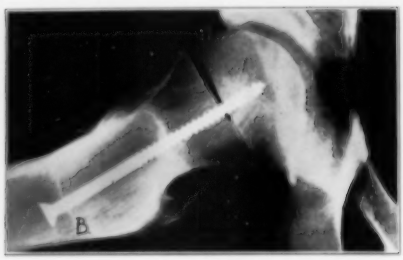

FIG. 14-B

Subcapital fracture. Symmetrical head in 90 degrees of anterior rotatory malposition. Fig. 14-A: Anteroposterior view. Trabecular pattern of head and neck do not match at fracture line. Fovea is not seen. Fig. 14-B: Lateral view. Trabeculae of head and neck not anatomically related at fracture line. Fovea seen in posterior part of head fragment.

FIG. 15-A

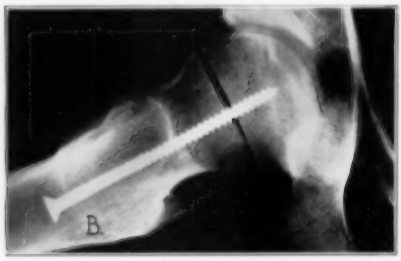

FIG. 15-B

Subcapital fracture. Symmetrical head fragment in 180 degrees of posterior rotatory malposition. Fig. 15-A: Anteroposterior view. Stress trabeculae belonging in lower half of head fragment shown in upper half and unmatched with trabecular pattern across fracture line. Fovea in superomedial part of fragment. Fig. 15-B: Lateral view. Same unmatched trabecular pattern at fracture line. Fovea not visualized.

site initially, or later, to jeopardize the circulation to the head will qualify the final result.

Normally, the circulation to the intact femoral head is made up of three groups of vessels: the intra-osseous vessel system, the retinacular vessels (anterior, superior, and inferior), and the foveal vessels through the ligamentum teres (Fig. 12, *A*). A linear subcapital fracture without impaction or displacement will interrupt only the intra-osseous blood supply to the head. If the head fragment remains undisplaced the retinacular and ligamentum teres blood supplies may remain unchanged and the outlook should be good (Fig. 12, *B*).

If a subcapital fracture is impacted in valgus position, the prognosis is usually good. The results seem to depend on what happens to the superior retinacular and ligamentum teres blood supplies. If the fracture occurs distal to the entrance of the superior retinacular vessels and if the degree of valgus deformity is not too great, both sources of supply may remain intact and the outlook should be good (Fig. 12, *C*). If the impaction occurs at, or proximal to, the entrance of the superior retinacular vessels, this source of blood supply is lost.

Most of our problems arise in management of the unimpacted symmetrical free femoral-head fragment. Rotatory movements of the head at the time of trauma, or by later handling of the fracture, can tear all retinacular supply to the head. If rotation is continued far enough the resulting twisting of the ligamentum teres will interrupt its blood supply (Fig. 12, *D*).

It would seem that not enough attention has been given to the effects which abnormal degrees of rotatory movement of the free femoral head may have on its own blood supply. It is the contention of this study that the rotational movements of the femoral head can be as destructive to its blood supply as any other

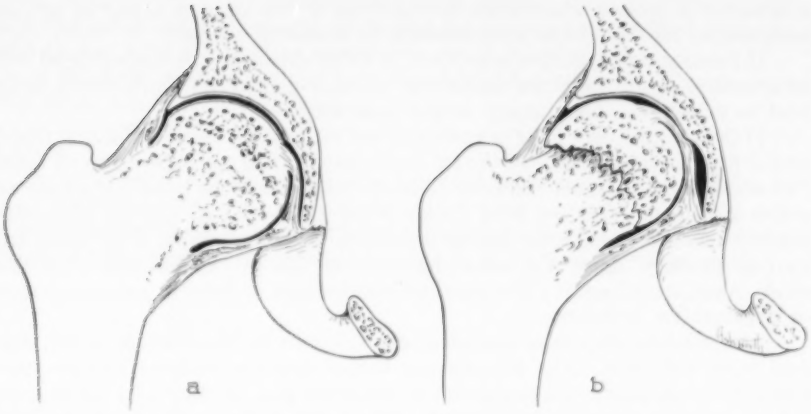

FIG. 16

A: Coronal section of intact hip showing normal relationship of ligamentum teres to acetabular fossa. *B:* Similar view of subcapital fracture with head fragment in extreme valgus position. Ligamentum teres insertion has left fossa superiorly, is overstretched, and compressed between lunate surface of acetabulum and femoral head. This position, from initial impaction or elective nailing, could result in avascularity of the head.

form of displacement. Assuming this is true, how can one determine at the time of reduction whether or not rotatory malposition is present? This might be done in one of two ways: open reduction preceding, or associated with, hip-nailing, and roentgenographic examination.

For obvious reasons, routine open reduction is not desirable. On the other hand, the information available from roentgenograms is limited. Routine anteroposterior and lateral roentgenograms of the fracture site, made either preoperatively or after reduction, properly assess apposition and alignment as well as angulation deformity. However, such roentgenograms usually do not demonstrate the presence or absence of rotational deformity of a symmetrical head fragment.

To illustrate this point, a hip specimen, denuded of all soft tissue, was artificially fractured to create a subcapital fracture with a symmetrical head fragment. The femoral head was then anatomically related to the femoral neck and transfixed by a wooden screw passed through the central neck from the lateral upper portion of the femur. By slight back-turning of the transfixation screw the head could be loosened from the neck and placed in various degrees of rotatory malposition.

Anteroposterior and lateral roentgenograms were made to show the hip in complete anatomical reduction with no rotation of the head (Figs. 13-A and 13-B). Similar views were made with the symmetrical head fragment in 90 degrees of rotatory malposition (Figs. 14-A and 14-B) and also in 180 degrees of rotatory malposition (Figs. 15-A and 15-B).

The details of bone structure in these roentgenograms are unusually good. This, of course, is due to the fact that there is no interposition of soft tissue. The loss of continuity of trabecular-bone pattern across the fracture site, together with changing positions of the fovea, demonstrate the presence and approximate the degree of rotatory malposition of the head fragment.

As these roentgenograms show, it is possible for a hip to be nailed in various degrees of rotatory malposition despite apparent anatomical relationship of head and neck. Depending on the direction and degree of this rotation, circulation to the femoral head could be affected (Table I).

Once a capital fragment is fixed to the femoral neck in rotatory malposition, the effect of subsequent hip movements on the ligamentum teres is changed. Here

a situation is created wherein the foveal blood supply may be active in certain positions of hip movement and deficient or absent in others.

If rotational malposition is extreme, in either direction, the femoral head will be avascular regardless of the position of the hip—a situation which would likely lead to non-union and primary aseptic necrosis.

If the rotation deformity is moderate, the subsequent course of healing might well depend on the positions of the hip maintained during convalescence and later. For example, in moderate posterior rotatory malposition one could anticipate an active blood supply to the head fragment during the initial months of recumbency, when the hip is in the extended position most of the time. This would encourage primary union. Yet subsequent uses of this hip in positions of flexion might result in ischaemia; this could ultimately lead to delayed aseptic necrosis or degenerative arthritis.

Malpositions other than rotational also influence the blood supply to the femoral head. Of these, only the extreme valgus position deserves further comment. Although slight valgus position in reduction may be desirable, the extreme valgus position should be avoided. In this position the insertion of the ligamentum teres leaves the acetabular fossa superiorly and, with its vessels, becomes compressed between the lunate surface of the acetabulum and the femoral head (Figs. 2, 11, and 16).

TABLE I

Type of Deformity	Effect on Circulation in Various Positions of Hip Movement
Extreme rotatory or valgus malposition	Complete interruption of blood supply
Moderate posterior rotatory malposition	Active blood supply on extension; slowed or interrupted blood supply on flexion
Moderate anterior rotatory malposition	Active blood supply on flexion; cessation of blood supply on extension
Moderate valgus	Active blood supply in abduction; slowed or interrupted blood supply in adduction

From the observations presented, it would seem that rotatory malposition and extreme valgus position might have considerable clinical significance. If this is so, their detection and correction should be added to our criteria of management.

SUMMARY AND CONCLUSIONS

Fifty-six open hip operations of different types afforded an opportunity to study the blood supply to the femoral head. In forty-four instances the ligamentum teres was cut; 70 per cent bled actively. Twenty-four capital fragments were examined *in situ* for the presence of active bleeding from their cancellous surfaces. Four were avascular, twelve bled sluggishly, and eight bled actively.

The eight heads were studied for the effects of various malpositions on their bleeding activity. Rotatory malposition, either anterior or posterior, interrupted active bleeding from the head fragments. Of the non-rotatory malpositions, only the extreme valgus position consistently interrupted bleeding.

Related anatomical studies were carried out on fresh cadaver specimens to determine the effects of normal and abnormal ranges of femoral-head movement on the ligamentum teres and its foveal vessels. The conclusions are:

1. In the intact hip it is impossible for the ligamentum teres to become sufficiently twisted, stretched, or compressed to interrupt its supply of blood to the femoral head.

2. In the fractured hip abnormal degrees of rotation of the free femoral fragment can easily result in twisting, stretching, and compression of the ligamentum teres to the point of complete interruption of its blood supply.

3. Rotatory malposition may be present and yet not be noticed in an otherwise anatomically reduced fracture.

4. Extreme valgus position interrupts foveal blood supply and should be avoided.

5. Fixation of subcapital fractures in either rotatory malposition or in extreme valgus position could account for many instances of delayed union, nonunion, aseptic necrosis, and degenerative arthritis. If this is so, detection and correction of these malpositions should be added to our criteria of management.

Congenital Muscular Torticollis in Infancy

Some Observations Regarding Treatment

BY MARK B. COVENTRY, M.D., AND LLOYD E. HARRIS, M.D., ROCHESTER, MINNESOTA

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The clinical entity seen in the infant and involving the sternocleidomastoid muscle, and generally termed congenital muscular torticollis, has been well recognized for centuries. Soon after birth a mass appears in the belly of the sternocleidomastoid muscle (Fig. 1). This is accompanied by a tilt of the head to the side of the muscle involved and by a rotation of the head so that the chin points to the opposite shoulder. After a time this muscle mass or tumor disappears. The mass and the tilt and rotation of the head and neck are accompanied by asymmetry of the skull and facial bones. This asymmetry tends to lessen and in fact disappear as time goes on, unless the wryneck becomes a permanent deformity.

The pathogenesis of this condition is obscure. All that can be definitely said at present is that it is probably not due to muscle tearing or hemorrhage at birth and that it is therefore not a birth injury *per se*. It is a well known fact that there is a high frequency of breech presentation of infants with congenital muscular torticollis. Lidge and associates in a recent extensive review of the literature concerning the etiology and pathology of congenital muscular torticollis cited eighty-seven references on this phase of the problem. Intriguing though investigation of the pathogenesis may be, unless it provides some means of prevention or an improvement in methods of treatment, it is perhaps of less practical use than the critical evaluation of treatment in a closely followed group of patients for whom both operative and non-operative methods are used.

Certainly the diagnosis of congenital muscular torticollis in infancy should be readily made by any informed physician today. But what form of treatment should be employed? There is no agreement whatsoever as to what constitutes

* The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

satisfactory treatment of this condition. This is perhaps due to the fact that since the pathogenesis is obscure the rationale for treatment also remains obscure. Available are physical therapy measures such as stretching and massage, or surgical measures which consist in either section of the sternocleidomastoid muscle, removal of the tumor itself, or a combination of the two. Many writers have advocated non-operative or conservative treatment. Chandler and Altenberg have perhaps been the most ardent exponents of operative treatment. In discussing this problem they, of course, distinguished between section of the sternocleidomastoid in older children who have not recovered from the initial congenital muscular torticollis and who have a contracted sternocleidomastoid and a wryneck deformity, and treatment of muscular torticollis of infants with a tumor of the sterno-

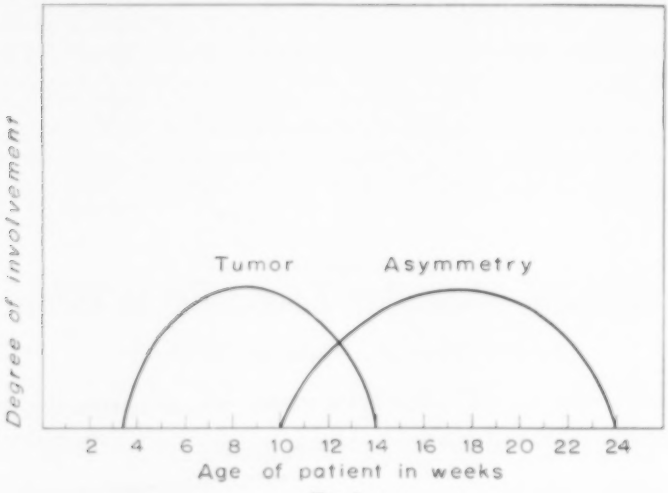

FIG. 1

Extent of involvement by sternocleidomastoid tumor and extent of asymmetry of face and skull according to age of patient.

cleidomastoid muscle. Chandler stated, "In view of the satisfactory results obtained by surgery in older children, it is debatable whether or not infants should be subjected to operation. In our cases only those who present the most severe contractures and the most extensive 'tumors' have been operated upon. We believe, however, that if we can prevent a deformity, rather than await its development, surgery is indicated. Surgical judgement must be exercised in its highest degree in the selection of cases. Our results have been so uniformly good and without complications that we lean towards the surgical approach to the situation, possibly more than may seem justified."

Chandler's view leaves the orthopaedist and the pediatrician in a dilemma, for nowhere does he mention just what criteria should be applied relative to "Surgical judgement must be exercised in its highest degree in the selection of cases." In other words, how is one to know upon which patient to operate? Are only the patients with the larger tumors to be selected for operation? How can one predict which infant will recover without operation? The present study was made in an effort to resolve this dilemma, at least in part.

MODE OF STUDY

From January 1, 1944, through December 31, 1954, thirty-five infants with congenital neonatal muscular torticollis were examined at the Mayo Clinic. Thirty of these infants were born in Rochester, Minnesota. Five were born in nearby areas but were seen by us shortly after birth. They were all examined by a pediatrician

on the first and fifth days of life and then at regular monthly intervals in the well-child clinic. When the sternocleidomastoid tumor was found in each of these infants, an orthopaedist was consulted. Each patient had, in addition to the tumor, some degree of tilt of the head and asymmetry of the skull and facial bones. Data on the mother's pregnancy and the delivery, including type of presentation, were noted. We examined thirty of the thirty-five patients one year or more after birth. The remaining five patients moved away and were examined by physicians in their home communities and the data of the follow-up examination were transmitted to us by these examining physicians who, in all cases, were the fathers of the patients and former fellows of the Mayo Foundation.

During the years 1944 through 1954 there were approximately 7,835 infants born to residents of Rochester, Minnesota. The proportion of these infants followed during these ten years at the well-child clinic averaged 95.3 per cent. There were thirty sternocleidomastoid tumors in infants born in Rochester during this period, making the incidence of congenital muscular torticollis approximately 0.4 per cent.

TABLE I

SEX, BIRTH PRESENTATION, AND SIDE INVOLVED
Thirty-five Patients

	No. of Cases	
Sex		
Female	19	
Male	16	
Presentation		
Breech	14	(40%)
Vertex	21	(60%)
Side of sternocleidomastoid tumor		
Right (9 breech, 7 vertex)	16	
Left (4 breech, 14 vertex)	18	
Bilateral	1	

TABLE II

RESULTS OF TREATMENT

Therapy	Result	
	Excellent	Poor
Physical	24	
None	6	
Surgical*	3	2
Total	33	2

* Three had preliminary physical therapy; two had no preliminary treatment.

OBSERVATIONS

Nineteen of the patients were girls and sixteen were boys (Table I). The sternocleidomastoid tumor was discovered, on the average, 3.5 weeks after birth. Chandler and Altenberg stated that it occurs between birth and ten to fourteen days thereafter, and Hulbert said that it occurs about ten days after birth. In the computation of the average for our series, two patients were omitted; one of these was not seen until the age of four months, at which time the tumor was resolving, and the other was not seen until the age of three and one-half months, at which time the tumor was of considerable size.

The tumor disappeared at the average age of fourteen weeks (Fig. 1). In about half the patients it was first discovered by the examining physician at the approximate age of four weeks, and in the other half it was discovered by the mother when the child was two weeks old. Many mothers did not know of its

existence, which is proof that these tumors are often not seen or found by the parents and is reason for the lack of history of tumor in many of the patients seen in later life with classic wryneck deformity.

The gestational histories of the mothers of these thirty-five infants were not unusual. Sixteen mothers were multiparae and nineteen were primiparae. The presentation was breech in fourteen (40 per cent) and vertex in twenty-one (60 per cent). The breech presentations were left in eight infants and right in six. The vertex presentations were right in nine and left in nine, and the side was not known in three. The left sternocleidomastoid was involved in eighteen infants, the right in sixteen, and both sides in one. Of the breech presentations themselves, nine were associated with involvement of the right sternocleidomastoid, four with the left, and one with both muscles. This finding was just the opposite of that for the vertex presentations, in which the right side was involved in seven and the left in fourteen. Thus, in the breech presentations there was approximately a 2:1 preponderance of the right side over the left, but in the vertex presentations there was a 2:1 preponderance of the left over the right. We do not know the significance of these figures.

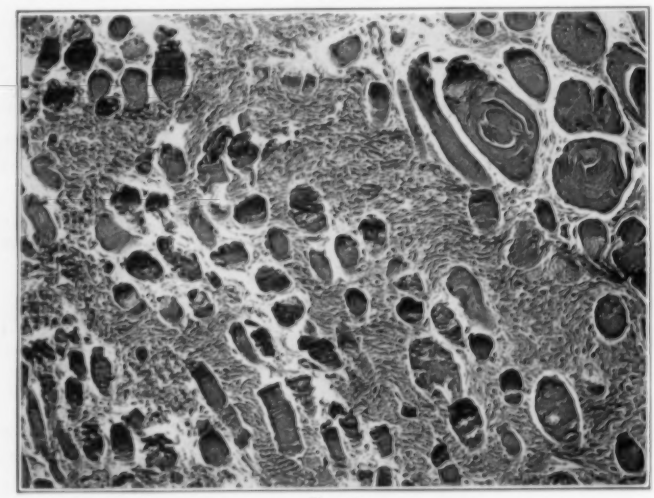

FIG. 2

Sternocleidomastoid muscle (hematoxylin and eosin, × 125). Note fibrosis interspersed among the muscle fibers.

The foregoing observations relative to sex, presentation, and side involved do not differ significantly from those of other authors; we have not, however, seen any reference elsewhere to the finding that the side involved varies with the type of presentation. Only two mothers had excessively traumatic deliveries; in one of these instances the obstetrician recorded a difficult vertex delivery and the infant had a subsequent brachial-plexus palsy; in the other the infant was delivered by breech and experienced a fracture of the clavicle at birth. The latter patient was the only one with bilateral congenital muscular torticollis. There were no associated congenital anomalies in the thirty-five patients except in one with possible dysplasia of the hip which was corrected completely with an abduction diaper splint.

Results of Treatment

As mentioned previously, the initial treatment of this condition can be directed either toward stretching and massage of the involved muscle or toward sectioning of the muscle with or without removal of the tumor. It should also be mentioned

that the condition can simply be observed, without active treatment of any kind being instituted. The patients who received physical therapy in this series were usually treated by the mother after instruction by a member of the Section of Physical Medicine and Rehabilitation. In some instances the pediatrician or the orthopaedist instructed the mother, and in a very few instances the patient received professional physical therapy in the Section of Physical Medicine and Rehabilitation for several weeks. It is obvious that the quality and quantity of physical therapy administered by mothers to their infants are extremely variable.

Results were graded as excellent or poor. An excellent result was one in which the patient had no remaining tumor, no apparent deformity of the neck, and no asymmetry of the face or skull. One may argue that all patients have some asymmetry of the face; we agree. But these children classed as having excellent results had no obvious or abnormal asymmetry a year or more after birth. A poor result was classed as one in which the patient had contracture of the sternocleidomastoid muscle with typical wryneck, that is, with head tilt, tight short muscle, and asymmetry of the face and skull.

All twenty-four patients who received physical therapy alone had excellent results (Table II). Six patients were not treated. They did not have lesser degrees of congenital muscular torticollis, but they were not treated because the pediatrician and the orthopaedist caring for them believed that they should be observed only. All six had excellent results. The remaining five patients were treated surgically. Physical therapy was first used in three; no preliminary treatment was given in two. Three of the five had excellent results, but two had postoperative recurrence of the deformity which required reoperation, and their results were therefore classified as poor. A closer analysis of the five surgical patients follows.

The first patient was observed to have the tumor at four weeks of age. Stretching was carried out without apparent improvement. At the age of four months, although the mass was much smaller, the muscle was tight and asymmetry was present. The sternocleidomastoid muscle was sectioned at the clavicular end, and the distal portion including the tumor was removed (Fig. 2). The head was immobilized in the overcorrected position for four weeks by a large felt dressing and bandage.

The second patient was observed at five weeks of age to have a tumor in the sternocleidomastoid muscle. The pediatrician and the orthopaedist were concerned about the exact nature of the tumor; as a result, excisional biopsy was performed when the patient was two and one-half months old. Study of microscopic sections confirmed the presence of a classic sternocleidomastoid tumor as seen in congenital muscular torticollis. No immobilizing dressing or physical therapy was used postoperatively. This infant had an excellent result.

In the third patient a tumor in the sternocleidomastoid muscle was diagnosed at one month of age. When the infant was three months old the muscle was sectioned at the clavicular end and the distal two thirds resected. Scar replacement of the lower two thirds of the muscle was seen. An immobilizing dressing in an overcorrected position was used postoperatively. An excellent result was obtained.

The fourth patient was observed to have a sternocleidomastoid tumor at three and one-half weeks of age. Physical therapy was instituted by the mother and her technique was checked frequently by a consultant in the Section of Physical Medicine and Rehabilitation. Stretching was found to be rather difficult because the baby cried and seemed, at times, to have difficulty breathing when the position of the neck was corrected. When the baby was six months of age, although the tumor had disappeared, the muscle continued to appear tight and the head tilted. The sternocleidomastoid tendon was sectioned at the sternal and clavicular heads, and the cut ends were allowed to retract. Apparent correction was obtained with this procedure, but at the age of four years the patient was called in for examination for the purpose of the present study. Wryneck deformity was found to exist. The roentgenographic appearance of the cervical part of the spinal column was

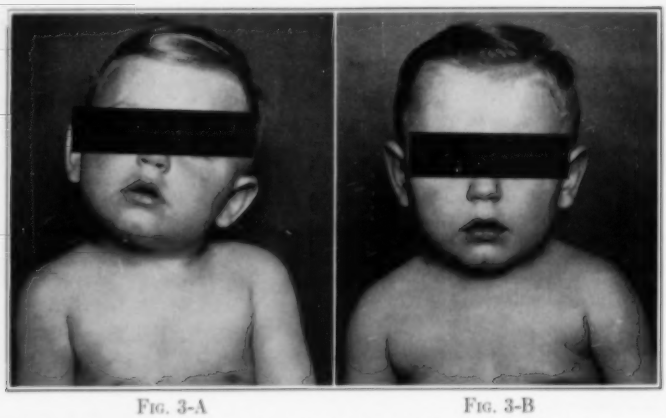

FIG. 3-A FIG. 3-B

Fig. 3-A: Before section of sternocleidomastoid muscle in Patient 5.
Fig. 3-B: Eight months after section of muscle.

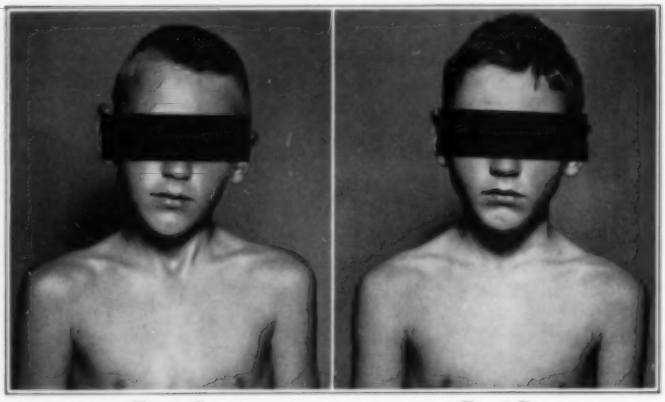

FIG. 3-C FIG. 3-D

Fig. 3-C: Tight sternocleidomastoid muscle with mild torticollis at the age of ten years.
Fig. 3-D: Three months after section of tight tendon and deeper structures.

normal. The deformity was due to contracture in the lateral portion of the sternocleidomastoid and in the platysma. The tight cervical fascia, the platysma muscle, and a few fibers of the remaining portion of the sternocleidomastoid muscle on the right were cut, and the sternoclavicular heads of the sternocleidomastoid on the left were sectioned as well. The surgeon thought this was indicated for cosmetic reasons because of the asymmetry of these tendons. Normal muscle and tendon were identified by the pathologist. The patient was last seen in 1958, three years later. There was slight tilt of the head to the right along with slight contracture in what appeared to be the platysma muscle. Further stretching and massage was advised. After the first operation, no immobilizing cast or dressing in an overcorrected position had been used, but active stretching had been encouraged soon after operation. The same procedure was followed after the second operation. At the first operation the muscle was sectioned near its attachment to the sternum and clavicle and the cut ends were seen to retract. At the second operation, sectioning was carried out more laterally.

The fifth patient was observed to have a tumor in the left sternocleidomastoid muscle at two weeks of age. Physical therapy was instituted at the age of one and

one-half months, much of which was given in the Section of Physical Medicine and Rehabilitation. It was considered adequate and was intensive. At the age of nineteen and one-half months, however, although the tumor had disappeared, there was still tightness in the sternoclavicular portion of the sternocleidomastoid muscle (Fig. 3). It was therefore elected to section the tendon at the sternal and clavicular insertions. The tendon was seen to separate for about one inch after being cut. Postoperatively the head was put in a traction sling and a sandbag was used to maintain the corrected position, but it was noted from the history that the patient was somewhat uncooperative and did not remain in the sling. He was given exercises postoperatively, but no immobilizing cast or dressing in the overcorrected position was used. When he was called back for examination, as a part of the present study, at the age of ten years, tight contractures were present in the left sternocleidomastoid muscle (Fig. 3-C). The skin, subcutaneous tissue, platysma muscle, and clavicular head of the sternocleidomastoid, together with the deep fascia and carotid sheath, were sectioned. A centimeter or so of the tendon was excised. Microscopic study showed tendon and fibrous tissue. A cast was applied postoperatively with the head in an overcorrected position and immobilization maintained for six weeks. The cast was then removed and active exercises encouraged. The result was excellent and has remained so, with no asymmetry or tilt of the head (Fig. 3-D). The sectioned muscle is, of course, smaller than the normal one.

In the years 1944 through 1947 we had fifteen patients with congenital muscular torticollis, four of whom were operated upon. In the years 1948 through 1954 we had twenty patients with congenital muscular torticollis, only one of whom was operated upon. This makes obvious our thinking regarding treatment of this condition. It is also obvious that we had no clear-cut criteria for the selection of patients for surgical treatment, and that our results do not indicate what would have been the fate of the surgical patients if they had instead been treated non-surgically.

SUMMARY AND COMMENT

This study of congenital muscular torticollis in infancy is continuing. The data obtained so far may be summarized as follows:

1. The tumor in the sternocleidomastoid muscle was discovered at an average age of 3.5 weeks and disappeared at an average age of three and one-half months. The longest period between time of appearance and time of disappearance of the tumor was five months. Hulbert gave the shortest period as two months, the longest as eleven months, and the average as seven months. Chandler gave the average period as five to eight months.

2. Asymmetry of the face and skull, although occasionally present at birth, was usually not evident until two and one-half months of age. Said in another way, facial asymmetry did not usually appear until about one and one-half months after the tumor appeared in the sternocleidomastoid muscle. The asymmetry tended to disappear for the most part, but as a rule not completely, by the age of six months. We did not detect any appreciable asymmetry in any of the patients with congenital muscular torticollis after one year of age, except in two whose results we have mentioned as poor. Asymmetry secondary to intra-uterine position is observed frequently at birth. This asymmetry is not permanent and has not been related to a subsequent development of a sternocleidomastoid tumor.

3. Two of the thirty-five patients have had residual wryneck. All others had excellent results. The two patients with poor results were among the five treated surgically. We do not infer that all patients will get excellent results, without wryneck deformity, if treated non-surgically; why these two surgically treated patients obtained poor results we can only guess. They were operated upon at six and nineteen and one-half months of age, respectively. No immobilizing cast or dressing in the overcorrected position was used. It is the impression of some [1] that

the overcorrected position should be maintained postoperatively to prevent reattachment of the sectioned muscle and tendon.

4. It is frequently said that no tumor exists in infants with well established wryneck, and it is even said that there may be two entities—one with tumor and one without. More accurately, it should be stated that there is no history of tumor in many patients with wryneck. Bianco found the incidence of wryneck without history of tumor to be about 80 per cent—only about 20 per cent of the patients with well developed wryneck deformity had a positive history of sternocleidomastoid tumor. Our studies throw some light on this point. Thus, as we have recorded, a sternocleidomastoid tumor was first found by the mother in 50 per cent of the infants in our study; in the other half it was missed by the mother, but was found by the physician during routine examination. Our study approaches the problem in the neonatal and early infancy periods and is based on examination of unselected infants; it is not a study based on infants brought to the physician because of tumor in the sternocleidomastoid muscle. We believe, therefore, that it presents an index of incidence, natural history, and results.

Statements [4,5] in the literature imply that as the neck begins to undergo relative lengthening at about the age of three years, the involved sternocleidomastoid fails to grow with corresponding rapidity, so that the onset of true wryneck occurs. Why does this occur in very few children? Why not in all? Is it a matter of degree of involvement by the tumor, that is, a matter of the ratio of normal muscle remaining to muscle replaced by fibrous tissue? Perhaps if enough normal muscle remains, it can grow, stretch, and otherwise adapt to the environment of growth and development, and no deformity will result.

Hulbert stated that four of every five tumors resolve completely and leave no deformity. In only two of thirty-five patients have we seen the tumor go on to produce deformity. When last examined, fifteen of our patients were one to four years of age, and twenty were four to twelve years of age; none of the twenty had wryneck deformity. It would seem from this number of traced patients that the incidence of development of torticollis after the tumor disappeared may be considerably less than the 20 per cent cited by Hulbert.

It is concluded that there is no justification for operating upon all patients with congenital muscular torticollis in infancy. If there is detectable wryneck deformity with contracture of the sternocleidomastoid muscle after the age of one year, this muscle should be sectioned then because the deformity at that age may be permanent. However, from our experience (Patient 5 after second operation) and that of others [7,8] there is every indication that an operation at any age up to twelve years will give as good a result as an operation at any earlier age. If the operation is properly carried out, there should be no residual deformity of the neck, except for atrophy of the sectioned muscle, which will occur if an operation is done at any age. The asymmetry of the skull and face will correct itself as long as growth potential is present. The child will grow and these bones will be reshaped in the absence of the deforming pull of a tight sternocleidomastoid muscle.

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Results of Treatment of Congenital Dislocation of the Hip*

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Beginning in 1942, some changes were introduced in the treatment of congenital dislocation of the hip at the State University of Iowa [7,8]. The period of immobilization in a plaster cast was reduced to a maximum of five months; usually three months in the first position, and six weeks or two months in the second position. This was followed by a prolonged period of functional after-treatment, controlled with an abduction bar or an abduction brace. At county medical society meetings the general practitioners, pediatricians, and obstetricians were instructed in the recognition of early clinical signs of this disorder. Early diagnosis made treatment of dislocation of the hip possible in the first years of life and often in early infancy. The results obtained from 1942 to 1950 in those patients who were initially treated before the age of four years are now reviewed.

The cases were classified in three groups: preluxations, subluxations, and dislocations. Hip preluxations were seen in infants under eight months of age and were characterized clinically by the snapping sign [6]. The displaced femoral head snapped into the center of the acetabulum by pressing the greater trochanter forward and abducting the thighs. In the roentgenograms the upper part of the femur appeared displaced upward or laterally (Figs. 2-A through 2-D).

In the group of hip subluxations were included children over six months of age with a limited range of hip abduction, but with a negative snapping sign. In the roentgenograms the small center of ossification of the femoral head appeared to rest under and lateral to the outer edge of the ossified portion of the sloping acetabular roof (Fig. 3). This deformity appeared to be more severe than the preluxation and could only be reduced after muscle relaxation achieved with either general anaesthesia or the application of an abduction brace or cast for several days or weeks.

The femoral head was shown to be above the margin of the ossified portion of the acetabular roof in the roentgenograms of patients with conditions classified as dislocations (Fig. 4). Hip abduction was often severely limited but the snapping sign was negative. General anaesthesia was required for the reduction of this advanced deformity.

Hip preluxations, subluxations, and dislocations represent stages in the development of congenital dislocation of the hip. During the first year of life no clear line of demarcation exists between preluxations and subluxations. Likewise, during the second and third years the distinction between severe subluxations and low dislocations may be difficult, even with arthrograms. Leveuf's contention [3] that these two conditions are unrelated is not supported by the extensive anatomical studies of Ortolani [4]. However, although some cases may overlap, the classification is useful for the selection of treatment and the evaluation of results.

After excluding cases of spina bifida, anterior poliomyelitis, and cerebral palsy, the records of seventy-eight patients with congenital dislocation of the hip were available. Nine patients had moved out of the state and could not be

*Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 28, 1959.

traced and five with arthrogryposis and teratological dislocation of the hips were excluded from the study. The remaining sixty-four patients were examined by the authors during the past six months, eight to sixteen years after initial treatment. Roentgenograms of the hips in neutral and Lauenstein's positions were made. This group included six patients with seven subluxations, fifty-four with seventy dislocations, and four with dislocation in one hip and subluxation in the other.

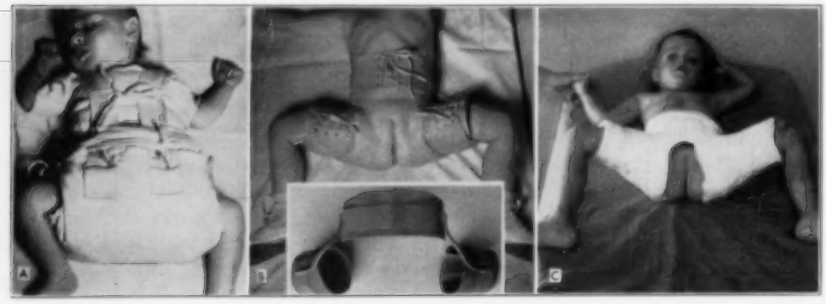

FIG. 1

The three types of bracing used in children initially treated prior to walking, showing: *A*, the Frejka pillow splint used in infants with mild hip preluxation; *B*, the abduction brace used in infants with hip preluxation and subluxation. The inset shows the posterior view of the steel frame and leather; and *C*, the hip spica plaster cast used in children with unilateral or bilateral hip dislocation or subluxation.

Thirty-two patients with preluxation treated during their first year of life, from 1942 to 1954, were also included in the study. In these cases, a four-year follow-up was thought sufficient to evaluate the results. The type of dislocation, sex, and side of involvement are classified in Table I.

TABLE I

TYPE OF DISLOCATION, SEX OF PATIENT, AND SIDE OF INVOLVEMENT

Classification	No. of Patients		Total No. of Patients	Involvement			Total No. of Hips
	Male	Female		Unilateral		Bilateral	
				Right Side	*Left Side*		
Preluxation	7	25	32	11	14	7	39
Subluxation	2	4	6*	4 (2*)	1 (2*)	1	11
Dislocation	6	52	58	18	24	16	74
Total	15	81	96	33	39	24	124

*Four patients with subluxation in one hip and dislocation in the other were included in the group of unilateral dislocations.

The snapping sign is the only unequivocal sign indicating that hip preluxation will develop into dislocation [6,13]. An examination for this sign was not routine procedure in our clinic until after the publication of Ortolani's book in 1948 [4]. This sign was positive in eighteen of the thirty-two patients with hip preluxation. Six patients with positive signs had bilateral involvement, and twelve had unilateral involvement. The sign was not recorded in the remaining fourteen, all of whom were seen before 1948. These patients were included in the study as preluxations because all were under six months of age, and aside from severe limitation of abduction, the upper portion of the femur appeared displaced upward or laterally in the roentgenograms [13] (Fig. 2-A).

The treatment used was outlined in previous papers [7,8]. However, to obtain a complete anatomical restoration of the joint, every case of congenital disloca-

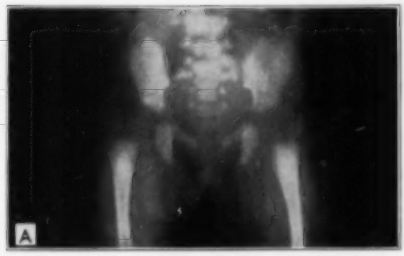

FIG. 2-A

FIG. 2-B

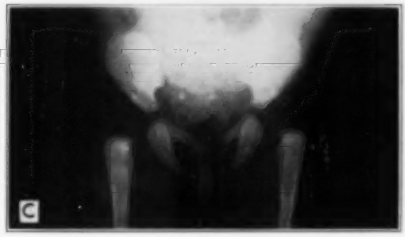

FIG. 2-C

FIG. 2-D

FIG. 2-E

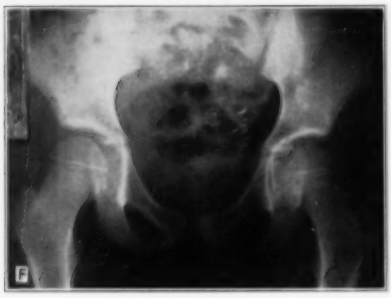

FIG. 2-F

Figs. 2-A through 2-F: Roentgenograms of hips with preluxation before and after treatment.

Fig. 2-A: Roentgenogram of the hips of a one-week-old female infant with preluxation of the right hip. The upper portion of the right femur is laterally displaced. Abduction was limited to 50 degrees in the right hip. The infant was not examined for Ortolani's sign. A Frejka pillow splint was worn full time for five months.

Fig. 2-B: Roentgenogram of the hips of a five-month-old female infant with severe preluxation of the left hip. Abduction was limited to 40 degrees in the left hip. Ortolani's sign was positive on the left. An abduction brace was worn full time for four months and at night and napping hours for an additional six months.

Fig. 2-C: Roentgenogram of the hips of a seven-week-old female infant with bilateral hip preluxation. Ortolani's sign was positive bilaterally. An abduction brace was worn full time for two months, followed by the application of a pillow splint for an additional three months. (Fig. 2-E shows the result at seven years of age. The CE angle is 22 degrees in both hips.)

Fig. 2-D: Roentgenogram of the hips of a five-month-old female infant with preluxation of the right hip. Ortolani's sign was positive on the right. An abduction brace was worn full time for two months, followed by the application of a pillow splint for an additional six months. (Fig. 2-F shows result at eight years of age. The CE angle is 20 degrees in the right hip and 22 degrees in the left.)

tion of the hip requires careful supervision, and some changes in the treatment may be necessary from time to time.

The anatomical results were classified into six groups in accordance with the criteria of Severin [10]. The use of the objective method of measuring the CE angle, as described by Wiberg [14] diminished the personal factor, especially in the cases which were most difficult to judge. The results obtained in the different types of dislocations are classified in Chart I. A well developed hip joint was

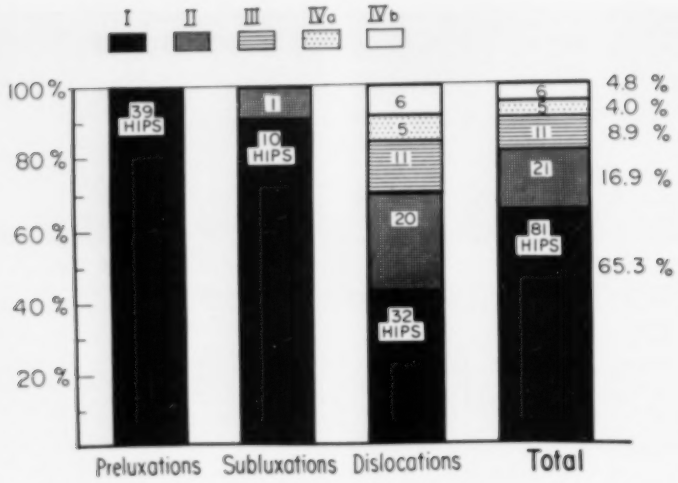

CHART I

Group I: Well developed hip joint. CE angle more than 20 degrees.
Group II: Moderate deformity of the femoral head, neck, or acetabulum in an otherwise well developed joint. CE angle more than 20 degrees.
Group III: Dysplasia, not subluxation. CE angle less than 20 degrees.
Group IVa: Subluxation, moderate. CE angle, zero or positive.
Group IVb: Subluxation, severe. CE angle, negative.
Group V: Femoral head in a secondary acetabulum in upper part of the primary acetabulum.
Group VI: Redislocation.

obtained in all preluxations and subluxations. Only 43 per cent of the dislocated hips developed well (Group I). However, the femoral head was in a correct position in the acetabulum in 85 per cent of the dislocated hips (Groups I, II, and III); subluxation developed in the remaining 15 per cent (Group IVa and IVb). No redislocations occurred and thus no results were classified in Groups V and VI.

The importance of early treatment is apparent when the cases are divided into three groups. Patients initially treated before one year of age are in Group A; Group B includes patients one to two years of age; and Group C includes patients two to four years of age (Chart II).

Group A: In Group A were included thirty-two patients with thirty-nine hip preluxations, six patients with seven subluxations, and eight patients with eleven dislocations.

The patients with hip preluxations—twenty-five unilateral and seven bilateral—were treated with an abduction brace worn for one to four months, followed by the application of a pillow splint worn for an additional three to six months (Fig. 1). A Frejka pillow splint worn for four to nine months was the only treatment used for twelve patients. All the patients had perfect anatomical results (Figs. 2-E and 2-F).

The patients with hip subluxations—five unilateral and one bilateral—were treated initially with a hip spica plaster cast maintaining the extremities in the first position for one to three months. The cast was applied with the patient under general anaesthesia in four instances and without anaesthesia in the other two. This was followed by the application of an abduction brace, worn for an additional two to five months (Fig. 1). The hips developed well in all these patients (Fig. 3).

The patients with hip dislocations—five unilateral and three bilateral—were treated by closed reduction while under general anaesthesia, followed by

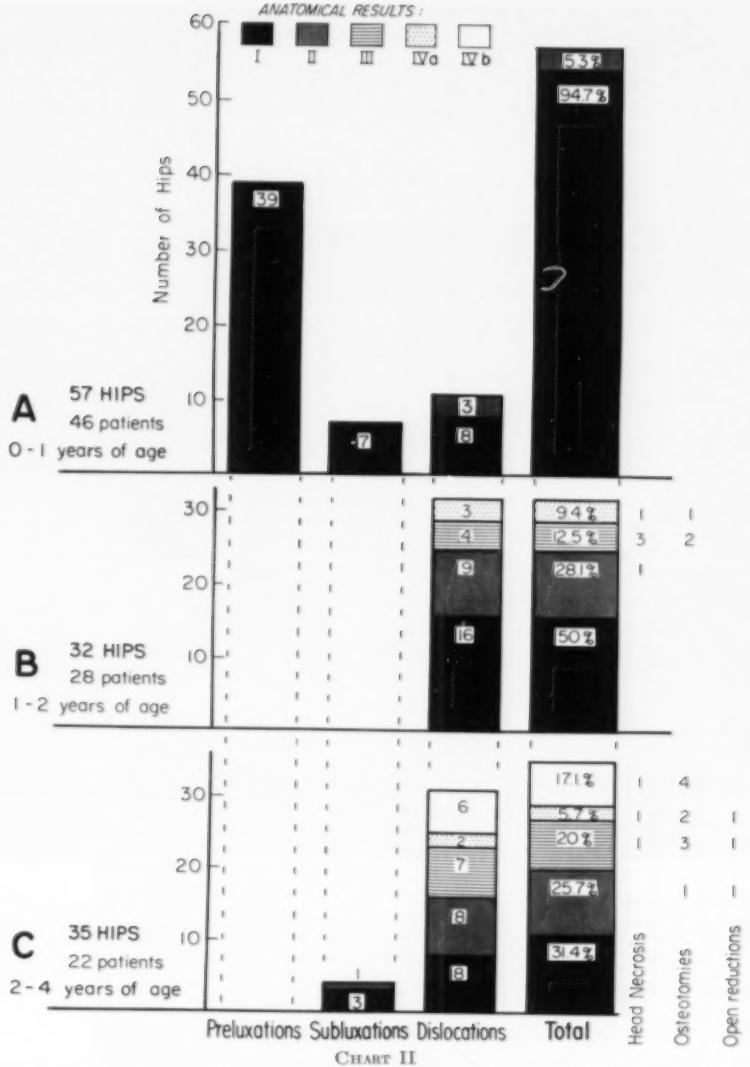

CHART II

The anatomical results according to age at initial treatment and the incidence of "epiphysitis" (necrosis of the femoral head), rotation osteotomy, and open reduction.

the application of a bilateral plaster hip spica with the extremities in the first position extending to just above the knees, worn for two to three months. An abduction brace was then worn full time for one to three months, and only at night and napping hours for an additional period of three to eight months (Fig. 1). Eight of the dislocated hips developed well (Figs. 4 and 5). A six-month-old girl with a superior prenatal dislocation of the right hip had slight coxa vara and the result was classified in Group II. A ten-week-old girl, the only Negro patient in our series, with a high bilateral prenatal hip dislocation and congenital genu recurvatum had a moderate deformity of the femoral head and

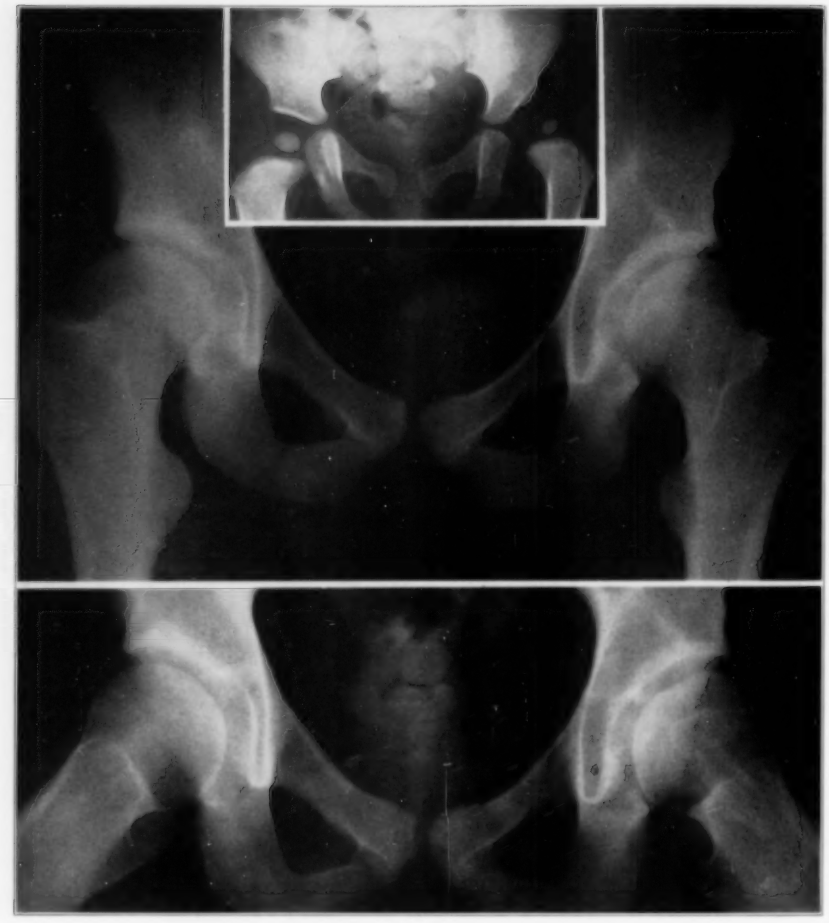

FIG. 3

Roentgenogram showing subluxation of the left hip (inset) in an eleven-month-old male infant. A bilateral short plaster hip spica with the extremities in first position was applied with the patient under general anaesthesia, and was worn for ten weeks. This was followed by the application of an abduction brace, worn full time for three months and at night and napping hours for an additional four months. The result (at seventeen years of age) was classified in Group I. The CE angle is 22 degrees in both hips.

neck of both hips (Figs. 6-A and 6-B). This result was also included in Group II.

Group B: Twenty-eight patients with thirty-two hip dislocations were one to two years of age when treated initially. Four patients had bilateral hip dislocation; the left hip was dislocated in fourteen, and the right hip in ten.

The dislocations were reduced with the patients under general anaesthesia. Preoperative skin traction for one week was used in only three patients with very high hip dislocation. Subcutaneous tenotomy of the adductor tendons was done on six hips. A bilateral plaster hip spica was applied, maintaining the extremities in the first position for two to four months with the thighs in 90 degrees flexion and about 70 degrees abduction (Fig. 7). The plaster cast extended to below the knee on the dislocated side for the first six to ten weeks and then was cut just above the knee, removing the lower section for the remaining period. After the closed reduction, soft-tissue interposition often prevented a complete

FIG. 4

Roentgenogram of a dislocated left hip in a seven-month-old female infant (inset). The dislocation of the hip was reduced with the patient under general anaesthesia, and a bilateral short plaster hip spica with the extremities in first position was applied and worn for three months. An abduction brace was then worn full time for two months and at night and napping hours for six months. The result (at thirteen years) was classified in Group I. The CE angle is 24 degrees.

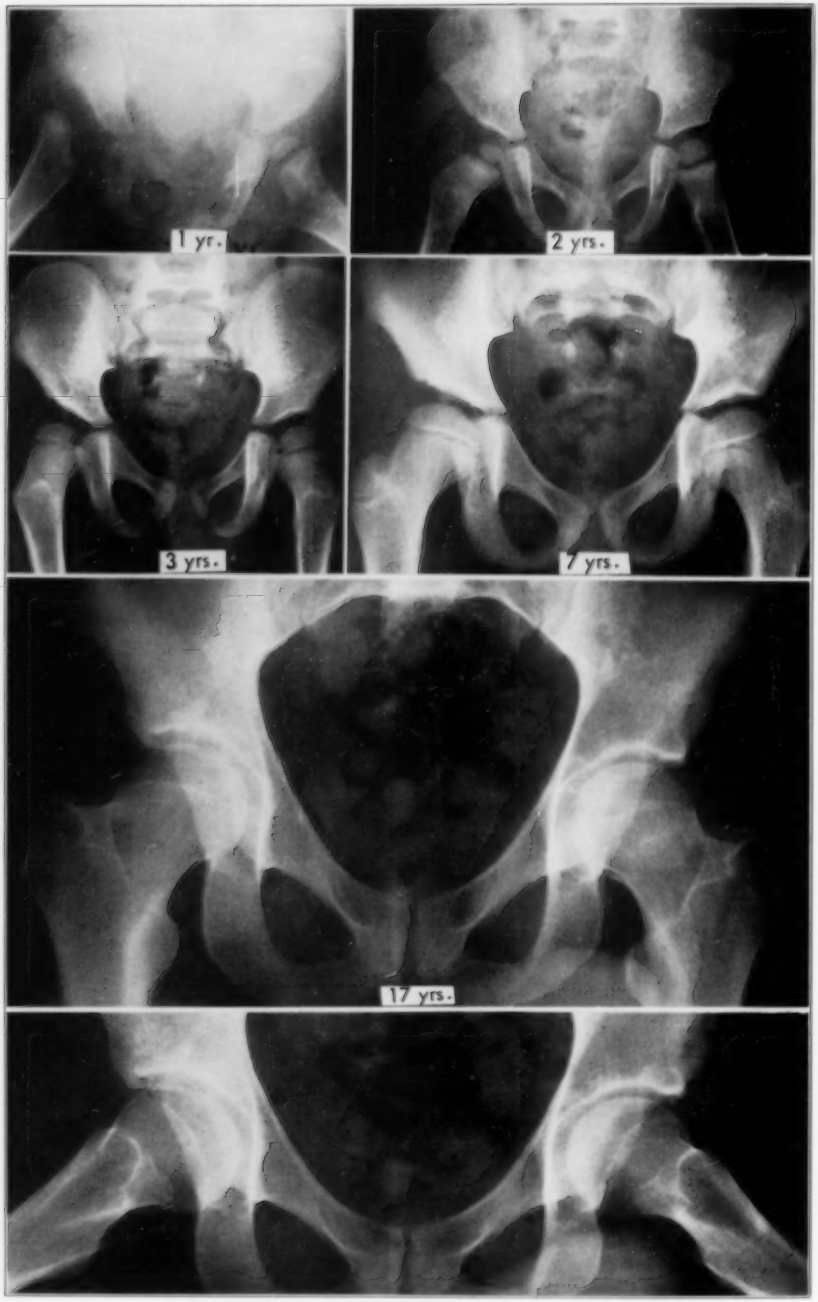

FIG. 5

Roentgenograms of the hips of a male infant with dislocation of the right hip and bilateral club-foot. The dislocation of the right hip was reduced with the patient under general anaesthesia at one year of age, and a bilateral short plaster hip spica was applied and worn for ten weeks. An abduction brace was then worn full time for three months and at night and napping hours for eight months. The acetabular roof developed very slowly. The result (at seventeen years of age) was classified in Group I. The CE angle is 34 degrees in both hips.

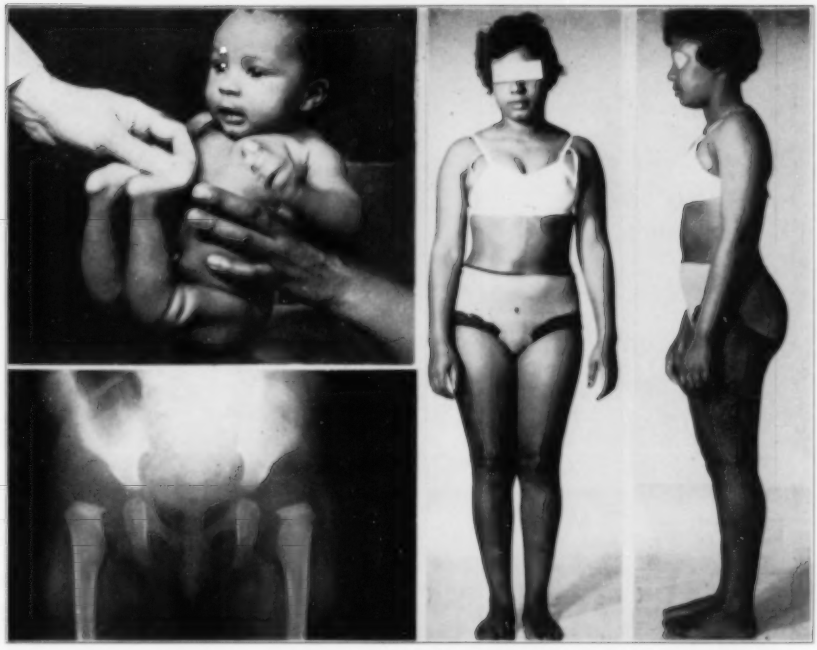

FIG. 6-A

FIG. 6-B

Figs. 6-A and 6-B: Severe bilateral hip prenatal dislocation and genu recurvatum in a female Negro infant. The knee deformity was treated by the application of three successive plaster casts, changed weekly, bending the knees gradually into flexion. The dislocations were reduced at ten weeks of age with the patient under general anaesthesia, and a bilateral long plaster hip spica with the extremities in first position was applied and worn for three months. An abduction brace was then worn full time for three months and at night and napping hours for six months. The result (at fifteen and one-half years of age) was classified in Group II. The CE angle is 20 degrees in the right and 27 degrees in the left. Coxa vara and a slight deformity of the femoral head were present in both hips. The girl was clinically normal.

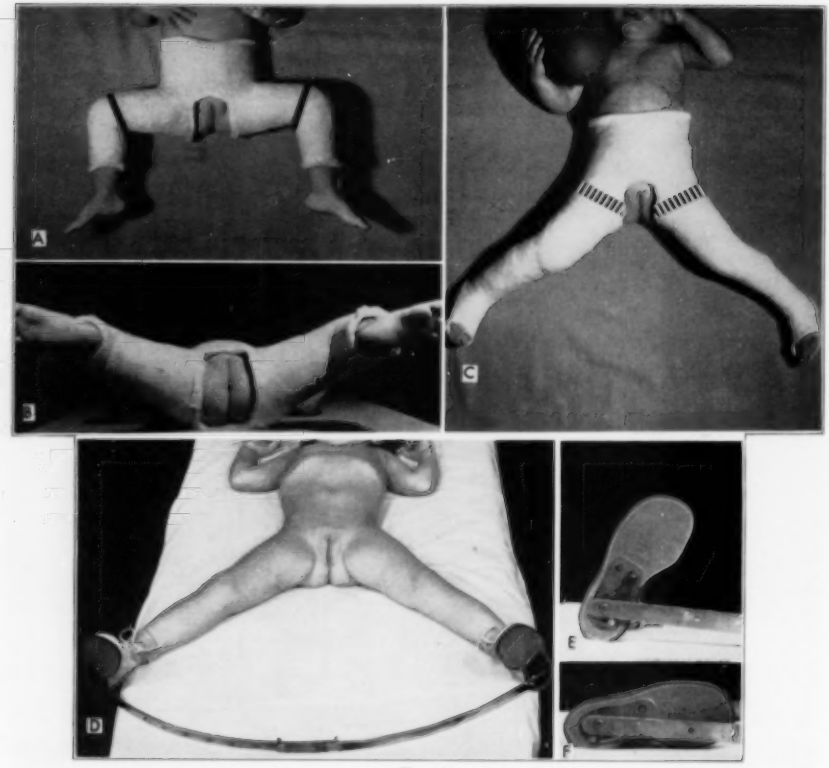

FIG. 7

Treatment of congenital dislocation of the hip in children over one year of age showing: *A* and *B*, a hip spica cast with the extremities in the first position applied after closed reduction with the patient under general anaesthesia. The plaster cast extends to below the knees in bilateral dislocation, and is cut above the knees (black line) removing the lower section six to ten weeks after reduction. The cast is removed two to six weeks later and the extremities are brought into the second position without anaesthesia; *C*, a hip spica cast with the extremities in second position. Two weeks after the change of position, a circular segment of the cast is removed at the hips (striped bands) to increase the inward rotation of the extremities. The cast is worn for one to two months; *D*, an abduction bar applied; *E*, outward rotation of the foot locked at 20 degrees short of neutral; and *F*, the inward rotation is free.

penetration of the femoral head into the socket. This soft-tissue interposition disappeared in a few weeks of treatment in the first position [11]. After removal of the first plaster cast the extremities were brought into the second position without anaesthesia, and a bilateral long hip spica was applied and then worn for one to two months. Two weeks after the change of position it was necessary, in most cases, to cut the cast at the hips to increase the inward rotation of the limbs. The femoral head remained well centered with the legs in 25 to 40 degrees inward rotation. An abduction bar was then worn full time for two to three months, and at night and napping hours for a period lasting from six months to two years. The patients started to walk a few days after removing the abduction bar during waking hours. During this period a tendency to subluxation was observed in six patients, and the extremities were maintained again in the first position with an abduction brace (Fig. 1-B) for two to four months. Two of these patients—one with bilateral involvement—had rotation osteotomy performed to correct femoral-neck anteversion eighteen months to three years after the closed reduction (Chart II).

FIG. 8

Roentgenogram showing dislocation of the right hip in a thirteen-month-old girl (inset). The hip was reduced with the infant under general anaesthesia. A bilateral hip spica was applied with the extremities in the first position and worn for ten weeks, and then, with the extremities in the second position, worn for two months. An abduction bar was worn full time for three months and part time for one year. The result (at thirteen years of age) was classified in Group I. The CE angle is 35 degrees in the right hip and 33 degrees in the left.

One patient with bilateral dislocation and fourteen patients with unilateral hip dislocation had well developed hip joints and the results were classified in Group I (Fig. 8). Two patients with bilateral hip dislocation and five patients with unilateral hip dislocation had slight deformity of the femoral head or neck, and their results were included in Group II (Figs. 9 and 10).

The hips of three patients with unilateral hip dislocation and one hip in a patient with bilateral hip dislocation were dysplastic (Fig. 11). These four patients had, initially, very high dislocations and their results were included in Group III. In three, "epiphysitis" developed six to ten months after the reduction; the sloping of the acetabulum did not become completely corrected in the other patient. Supracondylar rotation osteotomy was done on two patients of this group, two to three years after closed reduction.

Moderate subluxation developed in the hips of two patients with unilateral hip dislocation and in one hip of a patient with bilateral hip dislocation (Fig. 12). These three patients, whose results were included in Group IVa, were immobilized in the first position for only two months and in the second position

FIG. 9

Roentgenogram of the hips of a female infant twenty-three months old with bilateral dislocation of the hip (inset). The hips were reduced with the patient under general anaesthesia. A bilateral hip spica cast was applied with the extremities in the first position and worn for three months, and then in the second position and worn for two months. An abduction bar was worn full time for three months and at night and napping hours for sixteen months. The results of both hips (at eighteen years of age) were classified in Group II because of slight deformity of the femoral heads. The CE angle is 37 degrees in both hips.

for one month to six weeks. Subluxation occurred shortly after the patients started to walk, and although the hips were placed again in the first position with an abduction brace for a period of two to four months, subluxation persisted. Supracondylar rotation osteotomy performed eighteen months after reduction on one of these patients failed to correct the subluxation.

Fragmentation of the center of ossification of the femoral head was observed in five patients five to twelve months following the reduction. Moderate deformity resulted in one and a major degree of flattening occurred in the others (Fig. 11).

Group C: Twenty-two patients were two to four years old when treated initially. Nine of these patients had bilateral hip dislocation. Four had subluxation in one hip and dislocation in the other. Nine patients had unilateral hip dislocation.

The treatment used in this group was the same as that outlined for the patients one to two years of age. Five patients with high hip dislocation were

FIG. 10

Roentgenogram (*A*) showing high upward dislocation of both hips in a female infant, nineteen months old. Skin traction was applied for nine days prior to closed reduction performed with the patient under general anaesthesia. A bilateral hip spica cast was applied with the extremities in the first position and worn for ten weeks and then worn in the second position for six weeks. An abduction bar was worn full time for three months and part time for an additional three months. Bilateral subluxation occurred three months after walking, and the extremities were maintained again in the first position with an abduction brace (Fig. 1-B) worn full time for four months. The abduction bar was then reapplied and worn full time for two months and at night and napping hours for eight months.

Roentgenograms (*B* and *C*) made when the girl was ten and one-half years of age. The results in both hips were classified in Group II because of moderate deformity of the femoral heads. The CE angle is 30 degrees in the right hip and 35 degrees in the left.

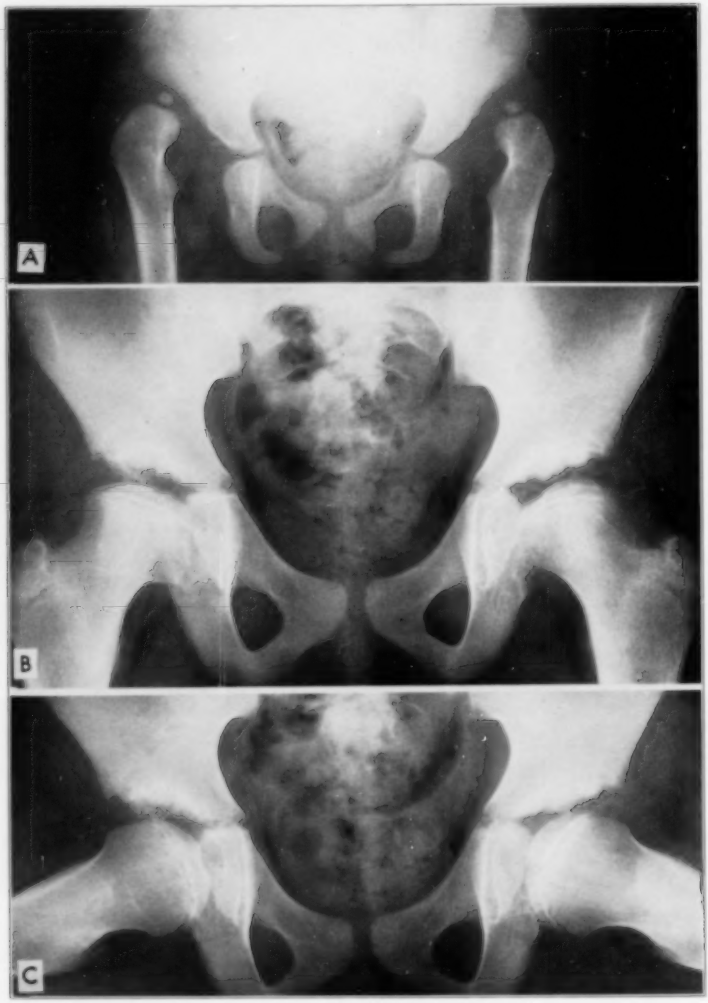

FIG. 11

Roentgenogram (*A*) showing high upward dislocation of both hips in a girl, two years old. The dislocations were reduced with the child under general anaesthesia. The reductions were difficult because no preoperative traction had been applied. A bilateral hip spica cast was applied with the extremities in the first position and worn for three months and then worn with the extremities in the second position for two months. An abduction bar was worn full time for three months and at night and napping hours for nineteen months. Necrosis of the centers of ossification of the femoral heads was apparent in roentgenograms made seven months after reduction.

Roentgenograms (*B* and *C*) made when the child was ten and one-half years of age. The results were classified in Group III because of severe flattening of both femoral heads and irregularly ossified acetabular roofs. However, the CE angle is 23 degrees in both hips.

treated for eight to twelve days in skin traction before reduction. Subcutaneous tenotomy of the adductor tendons was done on nine hips. Closed reduction failed, and open reduction was necessary in both hips of one patient with bilateral hip

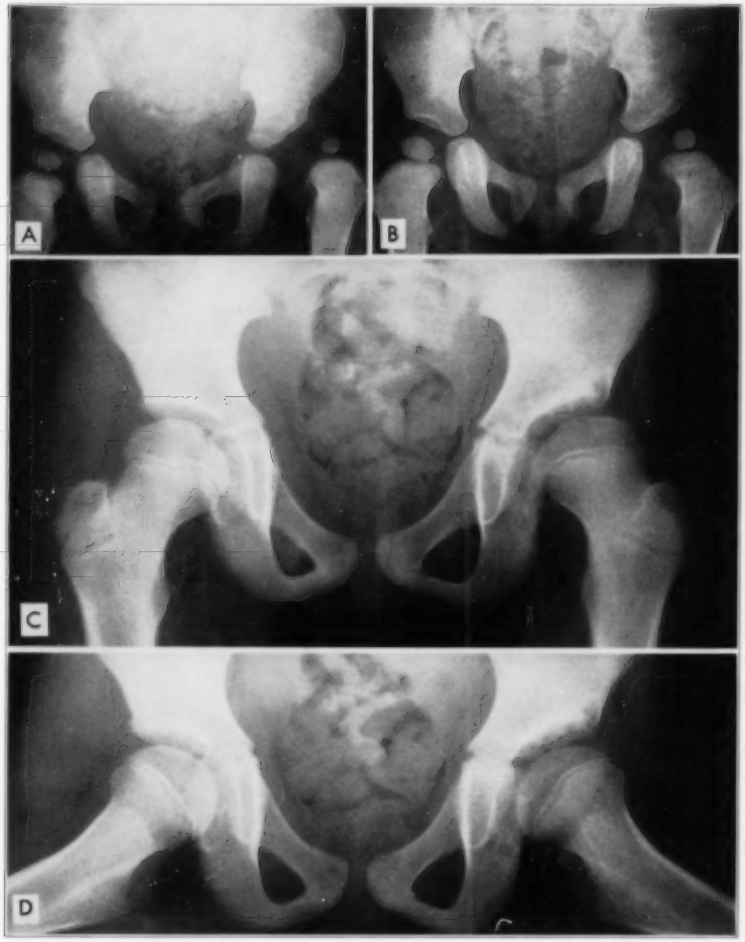

FIG. 12

Roentgenogram (*A*) showing dislocation of the left hip of a fourteen-month-old female infant. The hip was reduced with the patient under general anaesthesia. A bilateral hip spica cast was applied with the extremities in the first position and worn for only two months and then worn in the second position for one month. An abduction bar was worn full time for two months.

There was subluxation of the hip (*B*) one month after the patient started to walk. The extremities were again maintained in the first position with an abduction brace (Fig. 1-B) worn full time for three months and at night and napping hours for sixteen months.

Roentgenograms (*C* and *D*) made when the child was ten years of age show that a moderate subluxation persisted. The result was classified in Group VIa. The CE angle is 15 degrees in the left hip and 24 degrees in the right. The subluxation was probably due to the short period of immobilization in the first position.

dislocation and in the left hip of another patient with bilateral dislocation. Ten rotation osteotomies—six supracondylar and four subtrochanteric—were done to correct anteversion of the femoral neck from two to six years following reduction (Chart II).

In those patients whose results were classified in Group I (Fig. 13) well

FIG. 13

Fig. 13: Roentgenogram (*A*) showing dislocation of the right hip in a girl, three and one-half years old. The dislocation was reduced with the child under general anaesthesia. A bilateral hip spica cast was applied with the extremities in the first position and worn for three months and then worn with the extremities in the second position for two months. An abduction bar was worn full time for two months and at night and napping hours for eighteen months.

Roentgenogram (*B*) made when the patient was six and one-half years old, showing coxa vara and irregular ossification of the acetabular roof.

Roentgenograms (*C* and *D*) made when the patient was twelve years old. Coxa vara is almost completely corrected, and the acetabular roof is well developed. The result was classified in Group I. The CE angle is 30 degrees in the right hip and 26 degrees in the left.

developed hip joints were obtained in three hip subluxations and eight dislocations. There was moderate deformity of the femoral head, neck, or acetabulum in nine hips—one subluxation and eight dislocations. These results were classified in Group II (Figs. 14 and 15). A dysplastic hip was the result in seven hip dislocations. The dysplasia occurred in patients who were over three years old when initially treated and was due in three patients to defective formation of the acetabular roof (Fig. 14) and of the femoral head in three others. In one patient, coxa vara was the cause. One of the patients with defective formation of the acetabular roof had been treated by open reduction and rotation osteotomy. The two other patients with defective formation of the acetabular roof had rotation osteotomy performed which might have prevented subluxation.

The results in two patients were classified in Group IVa. Mild subluxation developed six months after operation in one hip treated by open reduction and rotation osteotomy. The other patient had a fragmented femoral head even before the closed reduction; neither the femoral head nor the acetabulum developed well (Fig. 16); rotation osteotomy was done two years after the reduction.

Severe subluxation developed in six hips from two to six years after satisfactory reduction (Figs. 17-E and 17-F). Rotation osteotomy, which did not seem to alter the end result, was performed on four of these patients.

Partial necrosis of the femoral head developed in three hips six to ten months after reduction. This resulted in some flattening of the femoral head in each instance.

DISCUSSION

The excellent anatomical results obtained in patients initially treated before the age of one year indicate the importance of early diagnosis in congenital dislocation of the hip. A positive Ortolani snapping sign indicates that the hip will dislocate and makes treatment imperative even in infants who are only a few weeks old. Roentgenograms usually show lateral or upward displacement of the upper end of the femur, but the roentgenograms may appear negative if they are made when the femoral head has snapped in the socket. A sloping acetabular roof and delay in the ossification of the femoral head is compatible with normal development of a hip joint in infants with a negative Ortolani sign. Infants with severe limitation of abduction of the hip and functional shortening of the femur probably should be treated with a pillow splint to avoid subluxation, although it is realized that the tightness of the adductors will correct spontaneously and the hips will develop well in many [1]. The patients with preluxation included in our study all had either positive Ortolani sign or definite roentgenographic disclosure of lateral or upward displacement of the femur, together with limitation of abduction of the hip. Most of these hips probably would have dislocated had not treatment been started early. Early treatment of congenital dislocation of the hip joint offers the only possibility of obtaining good results almost regularly [5]. Von Rosen and his orthopaedic staff, in collaboration with the pediatricians, discover all new cases of dislocations of the hip in newborn infants in Malmö, Sweden. Every orthopaedic surgeon should strive

FIG. 14

Roentgenogram (*A*) shows bilateral dislocation of the hip in a three-year-old girl. The dislocations were reduced with the child under general anaesthesia. A bilateral hip spica cast was applied with the extremities in the first position and worn for three months, and then worn with the extremities in the second position for six weeks. An abduction bar was worn full time for one month and part time for two years.

Roentgenograms (*B* and *C*) made when the patient was twelve years old. The result in the right hip was classified in Group II and of the left hip in Group III. The CE angle is 30 degrees in the right hip and 18 degrees in the left. The patient was clinically normal.

to this end in his community. The problems of congenital dislocation of the hip must be solved in the nurseries and not in the operating rooms.

Closed reduction, followed by carefully controlled treatment, gave good anatomical results in 78 per cent of patients initially treated when they were one to two years old (Groups I and II, Chart II, *B*). The unsatisfactory ana-

FIG. 15

Roentgenogram (*A*) shows bilateral dislocation in the hips of a girl, three and one-half years old. Skin traction was applied for six days. Closed reduction was successful in the right hip but failed in the left, which was surgically reduced. A very thick capsule constricting a deformed femoral head was found at operation. A bilateral hip spica was applied with the right extremity in the first position and the left extremity in the second position and worn for ten weeks. Both extremities were then immobilized in the second position in a hip spica worn for an additional month. An abduction bar was worn full time for two months and part time for fourteen months.

Roentgenograms (*B* and *C*) made when the patient was twelve years old. The result in the right hip was classified in Group I and that in the left in Group II. The CE angle is 25 degrees in the right hip and 30 degrees in the left.

tomical results were due either to necrosis of the femoral head or to mild subluxation (Groups III and IV, Chart II, *B*).

FIG. 16

Roentgenogram (*A*) showing dislocation of the right hip in a girl, twenty-six months old. The center of ossification of the femoral head is flat and irregular. The dislocation was reduced with the child under general anaesthesia. A bilateral hip spica cast was applied with the extremities in the first position and worn for only two months and then with the extremities in the second position for six weeks. An abduction bar was worn full time for ten weeks and part time for eleven months.

Roentgenogram (*B*) made when the patient was three and one-half years old. The right femoral head was irregularly ossified and not well centered. Rotation osteotomy was done when the child was four years of age. The subluxation persisted and at twelve years (*C* and *D*) of age the result was classified in Group IVa. The CE angle is 16 degrees in the right hip and 30 degrees in the left. The patient was clinically normal.

Only 57 per cent of the hips treated when the children were over two years of age had good anatomical results (Groups I and II, Chart II, *C*). Hip dysplasia developed in 20 per cent, and subluxation in 23 per cent. Deformity was severe in these patients and the opportunity for the normal development of a hip joint had passed. Only three open reductions were done when closed reduction failed (Fig. 15). A greatly thickened and constricted capsule adherent over an enlarged and deformed femoral head was found at operation. In all other

patients the head of the femur became well centered in the primary acetabulum following three months of immobilization in the first position. The poor results were due to defective formation of the acetabular roof or femoral head. In this group of patients subluxation occurred late, often two or more years after reduction (Figs. 17-A through 17-F).

The excellent results obtained in this series of patients in cases of preluxations and subluxations justify the continuation of treatment along the same lines. However, the results were not uniformly good in dislocated hips and here possible ways of improvement bear further scrutiny. The femoral head remained in a correct position in the acetabulum in sixty-three of the seventy-four dislocated hips (Groups I, II, and III); mild subluxation developed in five; and severe subluxation, in six. No redislocations occurred (Chart I). Except for three hips which were openly reduced, no difficulty was encountered in the reduction. The unsatisfactory results were seen in those hips with fragmentation of the ossification center of the femoral head and with late subluxation. The fragmentation of the epiphyseal nucleus was seen in patients with high dislocations not treated preoperatively with traction or in patients treated in the first position with the extremities in over 70 degrees of abduction (Fig. 11). This position presses the head of the femur against the acetabulum with great force and probably was responsible for the epiphyseal changes. Since 1950 we have avoided the extreme abduction of the extremities in the first position, and subcutaneous tenotomy of the adductors has been done when these muscles appear excessively shortened. In high dislocation traction before reduction may decrease the incidence of changes in the femoral head.

No proof exists that open reduction will decrease the number of late subluxations. Somerville and Scott[12] remove surgically the inserted limbus in patients with congenital dislocation of the hip. Our experience and that of Severin[11] and many others indicate that to obtain good reduction this is not necessary in the great majority of patients. Perhaps late subluxation may be prevented by the removal of limbus but we feel it advisable to wait for a longer follow-up period of the patients thus treated before judging the values of this operation.

This study indicates the necessity of maintaining the extremities in the first position for at least three months to center the femoral head and deepen the acetabular cavity. The hips treated in the first position for only two months had a tendency to subluxate. The second position followed by the abduction-bar treatment appeared to be useful in correcting femoral-neck anteversion. The child walks at first with the extremities in abduction and inward rotation; in this position the danger of early subluxation is lessened.

Rotation osteotomy performed to correct anteversion of the femoral neck prevented subluxation in few cases. Possibly the rotation osteotomy was done too late in some of our cases. However, the large number of good anatomical results in our series indicates that rotation osteotomy is rarely necessary in patients treated before three years of age.

We feel that some of the subluxations of cases in the old group could have been prevented by a shelving operation done before subluxation started to develop. In recent years we have performed this operation on selected patients and on all hips surgically reduced. The difficulty lies in establishing a definite indication for the shelving operation, since in the majority of our patients the acetabular roof developed well if the head remained well centered[9] (Fig. 5).

Most of the children of this series have been treated as out-patients. Their stay in the hospital was limited to a few hours after reduction. Anaesthesia was used only at the time of reduction. The child was free to crawl while wearing the plaster cast in the first position. During the last period in the first position, when the plaster cast was cut above the knees, the hips could rotate freely. When the extremities were changed into the second position, no anaesthesia was necessary. It was only during the six or eight weeks in the second position with

FIG. 17-A

FIG. 17-B

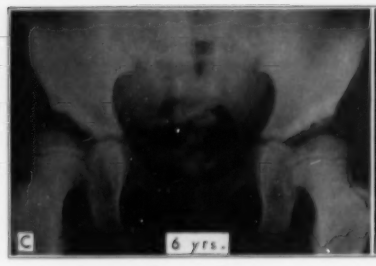

FIG. 17-C

FIG. 17-D

FIG. 17-E

FIG. 17-F

Fig. 17-A: Roentgenogram showing bilateral dislocation of the hip in a girl, twenty-five months old. The dislocations were reduced with the child under general anaesthesia. A bilateral hip spica cast was applied with the extremities in the first position and worn for three months and then worn with the extremities in the second position for two months. An abduction bar was worn full time for three months and at night and napping hours for twenty-two months.

Fig. 17-B: Roentgenogram made when the patient was four years of age. The acetabular roofs are shallow but the hips are not subluxated.

Fig. 17-C: Roentgenogram made when the patient was six years of age shows both hips to be slightly subluxated.

Fig. 17-D: The subluxations persisted at nine years of age.

Figs. 17-E and 17-F: Roentgenograms made when the patient was sixteen years of age show that the right hip is severely subluxated (Group IVb) and that the left hip is dysplastic (Group III). The CE angle is 16 degrees in the left hip and negative in the right (—15 degrees). The patient limped very slightly on the right, only when tired.

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a long leg hip spica that the activities were restrained. The child was little handicapped while wearing either the abduction brace or the abduction bar. Physical therapy was never used.

All patients had excellent functional results, except for six patients with severe subluxation who had slight limp and easy fatigability. Undoubtedly, in time, symptoms will appear in many more patients. An evaluation of the functional results cannot be done until several more years have passed.

SUMMARY

The anatomical results of treatment, from 1942 to 1950, of patients under four years old with congenital dislocation of the hip and of those with preluxation treated from 1942 to 1954 have been reviewed. Thirty-two patients with thirty-nine hip preluxations with a minimal follow-up of four years, six with seven subluxations, and fifty-eight patients with seventy-four hip dislocations and four subluxations with a minimal follow-up of eight years were included in the study. The dislocations were reduced with the child under general anaesthesia; only three open reductions were done when closed reduction failed. Rotation osteotomy was done on thirteen patients.

Good anatomical results were obtained in all cases of preluxation and subluxation (Groups I and II). Good anatomical results were also obtained in fifty-two of the dislocated hips; a dysplastic hip resulted in eleven instances. Thus, the femoral head was in a correct position in the acetabulum in sixty-three of the seventy-four dislocated hips. Moderate subluxation developed in five hips and severe subluxation in six. No redislocation occurred. Closed reduction followed by carefully supervised treatment offers satisfactory results in the majority of congenital dislocations of the hip if a child is seen before he is four years of age.

The importance of early treatment is apparent when the cases are divided into three groups. Good anatomical results were obtained in (a) all patients initially treated before the age of one year; (b) 78 per cent of patients who were one to two years of age; and (c) only 57 per cent of those who were two to four years of age. Early treatment offers the only possibility of obtaining good results almost regularly. The orthopaedic surgeon must collaborate with the pediatrician to discover all cases of congenital dislocation of the hip in early infancy. Ortolani's snapping sign is the only unequivocal sign of early dislocation.

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DISCUSSION

Dr. George E. Scheer, St. Louis, Missouri: Dr. Ponseti and Dr. Frigerio have demonstrated by the statistical review of their cases that early diagnosis and early treatment of dysplasia of the hip will give a high percentage of excellent results. I heartily agree with this conclusion and would strongly support the author's contention that it is imperative for all orthopaedic surgeons to bring the importance of early diagnosis and treatment to the attention of the pediatrician, the general practitioner, and even the nursing personnel.

During the first year of life, predislocations and subluxations constituted 80 per cent of the hip conditions seen and, in these, normal anatomical results were obtained. It is in the treatment of the dislocated hip after the first year of life that a result less than perfect is obtained. It is, particularly, in the group of children treated between the ages of two and four years that I feel that better than 57 per cent of the hips should have good anatomical results. In a recent review of a total of 135 cases of dislocation of the hip, prepared by Dr. Marshall Conrad, one of my associates at Shriners' Hospital in St. Louis, there were seventy-seven hips in children treated between the ages of two and four years. Of these seventy-seven hips evaluated from five to twenty-five years after completion of treatment, twenty-six (34 per cent) were classified as being normal, using the Gill method of classification; and thirty-three hips (43 per cent) of the total were classified as nearly normal. Thus, 77 per cent of the hips were either normal or nearly normal, from both an anatomical and a functional standpoint.

In addition, Dr. Ponseti and Dr. Frigerio reported eight children out of a total of sixty-seven in the age group of one to four years in whom necrosis of the femoral head was apparent. In reviewing our cases at the St. Louis Shriners' Hospital, in those patients in whom we had initiated the original treatment, we have had only one hip which we would classify as having had head necrosis. We feel that the significant differences are due to the fact that we (1) use preliminary skeletal traction, (2) do not perform manipulative reductions, and (3) do not immobilize the hip in a frog-leg position. Since contracture about a dislocated hip is a well recognized clinical finding we feel that this should be eliminated by preliminary skeletal traction prior to any attempt to reduce the dislocation. This permits the easy non-manipulative reduction of the hip and minimizes the compression force of the femoral head in the acetabulum after reduction. We also feel that although in the infant the functional position is flexion and, therefore, flexion positions are preferable, after the age of walking this is an unphysiological position and fixation in a position of extension, abduction, and internal rotation is preferred. Not only does the frog-leg position place the femoral head in the acetabular fossa in an upside-down position, but we have repeatedly demonstrated at open reduction that the head is more concentrically and deeply placed in the acetabulum in a position of extension, abduction, and internal rotation. Admittedly, this position could not be obtained easily without preliminary traction, but this is further support of the concept of preliminary skeletal traction.

We agree with the authors' statements that some of their subluxations may have been prevented by a shelving operation done before the subluxation started to develop. As the age of the dislocation prior to treatment increases, the less one can rely on the natural development of the acetabulum. Thus, we feel that acetabular reconstruction should be performed more often to provide support for the femoral head. Four loose criteria can be established for this procedure: (1) Age of the child, (2) a sharply inclined bony acetabular roof, (3) marked instability of the hip when tested under general anaesthesia and after preliminary skeletal traction, and (4) failure of the roof to develop rapidly after reduction.

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The Prenatal Development of the Skeleton and Joints of the Human Foot*

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INTRODUCTION

The early development of the upper and lower limbs has been described in recent publications [46,70,71,92]. Another recent report [28] deals with the prenatal development of the bones and joints of the hand.

Early Prenatal Skeleton

Various aspects of the prenatal development of the bones and joints of the foot have been studied by a number of investigators. The studies of Henke and Reyher [40] on embryos five to six weeks old were among the first to be published. These investigators reported that at this time all skeletal parts except some of the phalanges of the toes were laid down; the metatarsals and proximal phalanges were present, and the middle phalanges of the three lateral toes were present but indistinct. Henke and Reyher called attention to certain relationships of the tarsals and mentioned that the skeletal elements were separated from each other by what have since been termed homogeneous interzones.

Schulin [84] found that in a twenty-seven-millimeter embryo all skeletal elements except the distal phalanges of the toes and the sesamoids were present. Retterer [76] studied the feet of somewhat older specimens. He concerned himself primarily with the order of appearance of the ossific centers in the shafts of the metatarsals.

Lebouceq [55] reported that the youngest embryo in which the elements of the tarsus were differentiated was twelve millimeters long. In this specimen, the tibia and fibula were in contact, but the fibula lay at some distance from the tarsus. The calcaneus was separated into two parts by embryonic tissue. The sinus tarsi, present as a large space, contained a large blood vessel, which began to decrease in size in specimens larger than eighteen to twenty millimeters. In a later report, Lebouceq [57] stated that the distal phalanges were differentiated in the first half of the second month and that calcification at their tips began at the end of the second or the beginning of the third month.

Charles [15,16] described squatting facets on the tali and tibiae of Panjabi foetuses, infants, children, and adults, and questioned whether or not the squatting habits of the Panjabi led to the formation of these facets and their subsequent inheritance. Barnett [5], however, pointed out that, although rare in European adults, squatting facets are common in European foetuses. Presumably they disappear unless used. The theory of inheritance of acquired characteristics need not be invoked to explain their presence.

Several investigators studied chondrification in the foot. Hagen [29] observed the appearance of cartilage in metatarsals before tarsals. Schomburg [83] added further details of the sequence of chondrification in the foot. He noted that during

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the period of chondrification the fibula reached the calcaneus. Jones [51] confirmed the early relationship between the fibula and the calcaneus. Schomburg [83] claimed that the navicular arose from two centers in not quite half his specimens, that the medial cuneiform arose from two centers in more than half, and that the sustentaculum tali arose independently. Wilgress [103] described the early development of the lateral malleolus. Martin [62] noted chondrification in the metatarsals of fourteen-millimeter embryos. All phalanges except the distal phalanx of the little toe were chondrified by thirty-one millimeters.

Senior [86] was the first to analyze in detail the sequence of chondrification in the hand and foot. This sequence was confirmed by O'Rahilly, Gray, and Gardner [71], who related it to Streeter's "developmental horizons".

Hesser [42] paid special attention to the shapes of the articular surfaces. He found that the articular surfaces of the ankle joint reached a high degree of differentiation before a cavity was complete. The curvatures of some joints, such as the posterior talocalcanean, were still quite weak at forty millimeters but were beginning to resemble their final form. Early in the intermediate blastemal stage, the shapes of the talonavicular and calcaneocuboid joints assumed forms that agreed with their final shapes, whereas joints such as the intertarsal and tarsometatarsal were nearly flat, as in the adult, almost as early. The articular surfaces of the interphalangeal joints were differentiated earlier in the lateromedial than in the dorsoplantar direction. The dorsoplantar concavity on the articular surfaces of the bases of the distal phalanges was present before a similar concavity appeared on the bases of the proximal and middle phalanges.

Straus [91] stated that the joints of the foot were laid down in their definitive form early, certainly by the ninth week. He found that the medial malleolus was longer than the lateral in early fetal stages and the converse during later fetal and postnatal life. He observed changes in the shape of the talus during development and growth as well as differences in the growth of the phalanges, metatarsals, and tarsals. He noted that the hallux diverged from the other toes during early development and that this divergence was lost in later stages.

The general conclusion from the work of Hesser and of Straus is that the elements of the foot have shapes, arrangements, and relationships like those of the adult before the embryonic period (the first seven weeks after ovulation) is over and before cavitation begins. In a study of the first fourteen weeks of prenatal development, Harris [35] confirmed many of the results of previous workers and contributed a number of new findings. She found that the malleoli were not yet present in embryos at 16.5 millimeters but had become distinguishable by nineteen millimeters, when the tuber calcanei was also beginning to differentiate. She observed a shallow groove on the cuboid in embryos at 24.4 millimeters, and a groove for the flexor hallucis longus on the talus and calcaneus at 33.2 millimeters. Wilkinson [104] pointed out that the normal fibular deviation of the distal phalanx of the big toe is present in the human foetus; it was not found in other primates he examined.

Cartilage canals: Schomburg [83] observed the first appearance of cartilage canals in the talus and calcaneus. Hintzsche [43,44] noted cartilage canals in the calcaneus for the first time in foetuses at eighty-five millimeters and stated that they were abundant at ninety-two millimeters. Haines [31] studied the vascularization of cartilage during the fetal period and concluded that the canals arose by passive inclusion and were related to nutrition. Hurrell [47] also studied the vascularization of the tarsal cartilages, gave a concise review of the literature on this subject, and concluded that the vascularization was an active process. Harris [35] found cartilage canals in the calcaneus and talus in foetuses at 60.9 millimeters, in the cuboid and navicular at 80.5 millimeters, and in the cuneiforms and metatarsals at 107.2 millimeters.

Ossification: Schomburg [83] described the first appearance of bone in the various elements of the foot. The order of ossification was studied in somewhat more detail by Rückert [79], who used macroscopic, microscopic, and roentgenographic

methods, and also by Hasselwander [38]. Hintzsche [44] noted periosteal bone on the calcaneus in foetuses at 190 millimeters. Endochondral bone was present in the calcaneus of all specimens from six months on. Two ossification centers, periosteal and endochondral, were present in more than half of the cases. In a later report, Hintzsche [45] gave further details of the ossification of the calcaneus and paid special attention to the development of the markings on the lateral aspect of the calcaneus. The former feature has also been studied by Meyer [65]. Jit [49] used alizarin red S and studied prenatal ossification in the feet of Punjabi foetuses. He stated that bone in these elements seems to appear later than in the feet of foetuses in Western countries and that the later appearance may be owing to a nutritional basis. Hasselwander [39] published a summary of his own findings as well as of the findings of others on the ossification of the foot. He provided information on the sequence of diaphyseal ossification in the foot, most of which takes place prenatally, and on the sequence of epiphyseal ossification, which occurs after birth. Attention was called to the sexual difference in the ossification of the epiphyses and to the times of fusion of the epiphyses in both sexes. Prenatal ossification has recently been studied by Noback and Robertson [67], who used foetuses that had been cleared and stained with alizarin red *in toto*, and by O'Rahilly and Meyer [72], who investigated silverized specimens by means of roentgenography. Meyer [65] subsequently carried out a detailed study of prenatal ossification in the elements of the foot.

First metatarsophalangeal joint: In a study of the first metatarsophalangeal joint, Barlow [4] confirmed earlier reports by Leboucq [54] and by Straus [91] in regard to the divergence of the big toe. Barlow maintained that the divergence was owing to the obliquity of the joint surfaces and that the obliquity resulted from the unequal lengths of the two sides of the medial cuneiform. He stated that the divergence of the hallux is more marked up to seventy-five millimeters and that it becomes reduced by the growth of the medial side of the medial cuneiform. In connection with his study of the first metatarsophalangeal joint, Barlow noted that the beginnings of the transverse metatarsal ligament were present at thirty-two millimeters and that the tendon of the peroneus longus was not attached to the medial cuneiform until 135 millimeters. Jones [51] stated that the specifically human features of the big toe are completed by thirty millimeters.

Sesamoids: Detailed accounts of the prenatal development of the sesamoids of the foot are not available. Gillette [25] reported that they were present as cartilage in the foetus and the new-born infant and that they ossified late. Chemin [17] stated that he saw sesamoids in foetuses at two and a half months. Schomburg [83] noticed that they appeared later than the phalanges and that they did not occur in as large numbers in the foot as in the hand. He reported that two sesamoids were present regularly in the big toe, two in the small toe in about half the cases, and one in the latter toe in the remaining cases. He found a sesamoid for the first time in the tibialis posterior in foetuses at fourteen weeks and an interphalangeal sesamoid in the big toe first at fifteen weeks. Inge and Ferguson [48], in a study of the metatarsophalangeal sesamoids of the big toe, found that they consisted of undifferentiated connective tissue at eight weeks and of definite cartilage at twelve weeks; by eight years they were normally ossified, and by eleven years ossification was complete. Kewenter [53] also studied the metatarsophalangeal sesamoids of the big toe. However, his studies were by roentgenography and were confined to the postnatal period. He found that ossification began between seven years and two months and ten years and nine months in girls, and between nine years and ten months and twelve years and five months in boys. Kewenter also made observations on the position, shape, size, and divisions of these sesamoids. Boeminghaus [10] studied the development of the medial sesamoid at this joint and concluded that its occasional bipartite condition was the result of two centers of ossification. In a more recent study of the development of the sesamoids of the big toe, Takahashi [94] found that the lateral metatarsophalangeal sesamoid appeared at seventy-

five days, the medial at eighty-two days, and the interphalangeal at the fourth month. Harris [35] saw indications of sesamoids at the first metatarsophalangeal joint in foetuses at forty-three millimeters and definite cartilaginous sesamoids at 60.9 millimeters. In her series, an interphalangeal sesamoid first appeared in this digit at 101.8 millimeters.

Fusions and accessory elements: Fusions and partitions of normally occurring skeletal elements in the foot during the fetal period have been observed by a number of workers [35,38,56,61,75,90]. Descriptions of accessory cartilages in the foetus have been published by Bardeleben [2] and by Trolle [97]. O'Rahilly [69] gave a review of the literature on their presence in the adult.

Interzones, cavities, synovial tissue, capsules, ligaments, and tendons: The classic works on the development of interzones, joint cavities, and synovial membrane are those of Bernays [9], Schulin [84], and Hagen-Torn [30]. Studies dealing specifically with the development of the joints of the foot are more recent. Faldino [21], for example, found that at twenty-five millimeters the ankle joint showed signs of cavity formation and that at thirty-five millimeters a cavity was present in this joint. The interphalangeal cavities were the last to appear, and in his series all cavities were not present until 210 millimeters. Martin [62] reported that homogeneous interzones ("intermediate cell masses") were present in all joints of the foot at twenty-one millimeters. Fissures appeared in some joints at forty-eight millimeters and in most joints by fifty-one millimeters. Synovial tissue, containing many blood vessels, was present in a foetus of fifty-nine millimeters, and at 140 millimeters the synovial membrane contained abundant collagenous fibers. Vascular synovial folds were present at this time. Good photomicrographs of the fetal ankle joint were published by Barnett and Napier [6]. Martino [63] described the histology of the chondrogenic layers of the interzone and stated that they were fused at three months. Beau [7] found homogeneous interzones containing flattened cells at thirty-three millimeters. Fissures appeared at forty millimeters, and cavities were well developed at sixty-five millimeters. Synovial folds had appeared by seventy millimeters. In a study of the ankle joint, Carleton [13] claimed that a cavity was not yet present at ninety millimeters. Haines [31] summarized the earlier work on the development of joints and included personal observations. He found that the tarsal elements were chondrified at fourteen millimeters and that they were separated by dense interzones at this time. He observed that three-layered interzones first appeared in the ankle and talocalcanean joints at twenty-seven millimeters, and in the digital and smaller intertarsal joints at thirty-two millimeters. Harris [35] noted three-layered interzones at twenty-five millimeters, but she did not specify the joints in which they appeared. She found evidence of cavity formation at 33.2 millimeters in the ankle joint, and, at 37.4 millimeters, spaces were beginning to form in the ankle and in most of the tarsal and tarsometatarsal joints. By 60.9 millimeters, all joints showed evidence of cavity formation. The development of the capsules and ligaments of the joints of the foot has received somewhat less attention. Schulin [84] stated that in general the capsular insertions of all joints of the limbs were in their definitive places by the second half of the fourth month. Lucien [58] observed that the "anterior annular ligament" of the ankle joint appeared as a cellular condensation at thirty-three millimeters and that at forty millimeters this ligament had two extensions into the sinus tarsi. The ligament was fully developed at sixty-five millimeters. Lucien [59] also studied the development of the "lateral internal ligament" of the ankle joint and found that it appeared slightly earlier. Faldino [21] reported that capsules and ligaments were indicated at forty-one millimeters and that they were present in specimens of sixty-one, sixty-three, and sixty-five millimeters. Interosseous ligaments appeared by ninety-five millimeters. Thomas [95] described the arrangement of the tarsometatarsal ligaments in the foetus. Beau [7] described in detail various ligaments at different stages. Some ligaments were identifiable at thirty-three millimeters; the talotibial was the clearest of these, and it was fibrous. At forty millimeters, the capsules were

indicated, and ligaments had appeared in the sinus tarsi. Capsules were definitely present, although not yet very thick, at sixty-five millimeters. Beau mentioned that some ligaments appeared later as differentiations of the capsules. A study of the tarsometatarsal ligaments by Beau and Simonin [8] revealed that these ligaments were first indicated at thirty-three millimeters. They reported that the tendons of the tibialis posterior and the peroneus longus were also in their definitive positions at this time. Harris [35] found ligaments at twenty-five millimeters, but they were poorly defined. However, they were well developed at 27.8 millimeters and later.

Many of the discrepancies in the figures for the lengths from the crown to the rump given by different investigators for the time of first appearance of various structures in the embryo and foetus are attributable to the lack of preciseness of these lengths as an index of the stage of development. Streeter [93] made a very valuable contribution to the timing of events by dividing the embryonic period proper into twenty-three "horizons" or stages and by describing in some detail the degree of development of certain structures at each stage. The increasing use of Streeter's stages by investigators will undoubtedly lessen the confusion that now exists with regard to the times at which structures appear during the embryonic period.

Attempts to correlate foot lengths with crown-to-rump lengths and age by Retzius [77], Streeter [92], Meredith [64], Trolle [96], and Meyer [65] have met with some success. However, foot lengths are not as precise as the criteria for the staging of embryos described by Streeter. Therefore, the accurate timing of events that take place during the fetal period awaits furthur investigation.

MATERIALS AND METHODS

One hundred and eighty-four human embryos and foetuses were studied; they ranged from seven to 460 millimeters in length. The approximate ages of the embryos can be gauged from Streeter's [93] graphs; those of the foetuses from Plate 11 of Boyd's monograph [11].

The majority of the specimens were fixed in formalin. The larger foetuses were decalcified in a mixture of equal volumes of 50 per cent formic acid and 20 per cent sodium citrate. Paraffin, celloidin followed by paraffin, or celloidin alone were used for embedding, depending on the size of the specimen. Most of the embryos were sectioned *in toto*. The feet were removed from the remainder of the specimens and sectioned separately; in most cases the separation was effected through the middle or the lower part of the leg. Frontal, sagittal, and horizontal planes of section were used. Most sections were stained with a Mallory-Masson technique, but several other stains were employed.

The following list shows the lengths in millimeters of the specimens studied (the measurements of the older embryos and of the foetuses are crown-to-rump lengths):

7.0 (2), 8.1, 8.6, 9.0 (2), 9.7, 10.1 (2), 10.5, 10.8, 10.9, 11.0 (3), 11.2 (2), 11.5, 11.7 (2), 12.0 (2), 12.5 (2), 12.6, 12.8, 13.0 (4), 13.2 (3), 13.4, 13.8, 14.0 (3), 14.2, 14.4, 14.5, 15.0 (3), 15.5 (2), 16.0 (2), 16.5, 16.8, 17.0 (5), 18.0 (6), 18.5 (2), 19.0 (2), 19.1, 19.5, 20.0 (2), 20.8, 21.0 (4), 21.5 (2), 22.0 (3), 22.5 (2), 22.7, 23.0 (2), 23.4, 23.5, 24.0 (2), 24.2, 24.5, 25.0, 25.3, 26.0, 26.3, 26.4, 27.0, 27.3, 27.5 (3), 28.0 (2), 28.2, 30 (4), 31 (2), 32, 35, 35.5, 37 (3), 37.8, 39 (3), 43 (2), 45, 46 (2), 49, 50 (3), 55, 58, 60 (2), 61 (2), 65, 68, 69, 73, 78, 83, 85, 90, 93, 95, 100 (2), 104, 106, 107, 110, 117, 118, 122 (2), 124, 125 (2), 130, 132 (2), 136, 144, 145, 150 (2), 156 (2), 160, 180, 192, 195, 198, 235, 253, 262, 270, 285, 290, 302, 308, 348, 359, 373, 460.

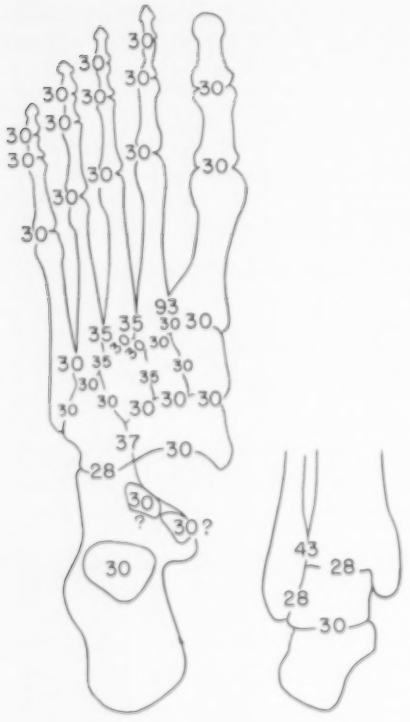
The figures in parentheses indicate the number of specimens studied of the same length.

One hundred and six of the embryos and foetuses were from the collection of the Department of Embryology, Carnegie Institution of Washington. The embryos

belonged to Stages 16 to 23. The writers are indebted to Dr. George W. Corner for permission to study these specimens.

Eight foetuses (61, 78, 93, 118, 122, 124, 125, and 150 millimeters) had been pretreated with silver [72], and had been used by Meyer [65] in a study of prenatal ossification.

The term *foot* as used throughout this paper includes the ankle, tarsus, metatarsus, and phalanges. The terminology employed is the *Nomina anatomica* of 1955, translated into English when appropriate.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blastemal in their distal parts. Areas of hypertrophied cartilage cells (Streeter's Phase 3) were present in the shafts of both, but those in the fibula were less advanced.

The lower ends of the tibia and fibula still consisted of condensed mesenchyme at Stage 20 (twenty-one to twenty-three millimeters), and the fibula was closely related to the calcaneus. Streeter's Phase 4 cartilage was present in the tibia but not in the fibula. By Stage 21 (twenty-two to twenty-four millimeters) Phase 4 cartilage was present in the tibiae of all embryos and in the fibulae of some. There were no bone collars, although osteoblasts were observed in the perichondrium of the tibia of some embryos. The fibula, which was closely related to the calcaneus, was separated from it by a densely cellular zone of flattened cells. In some specimens, the malleoli were beginning to chondrify.

A bone collar encircled the shaft of the tibia of most specimens at Stage 22 (twenty-five to twenty-seven millimeters). The fibula lacked a bone collar but contained Phase 4 cartilage.

At the end of the embryonic period (Stage 23, twenty-eight to thirty millimeters) the bone collar of the tibia was thicker and in one specimen was being eroded by periosteal buds. The fibula lacked a definite bone collar except in three specimens in which an early collar was present. The lower end of the fibula was still closely related to the calcaneus, but was usually separated from it by a small part of the talus.

Skeleton of Leg: Fetal Period

Vascular invasion of the shaft of the tibia, first noted in a thirty-millimeter embryo, was constantly present in specimens of thirty-seven millimeters and larger. Vascular invasion of the fibula was first present in a thirty-millimeter foetus, and in all specimens from forty-three millimeters onward. Endochondral ossification was generally present in the tibia from forty-three millimeters on, and in the fibula from fifty millimeters.

A cartilage canal was found in the lower end of the tibia at sixty millimeters, and in all specimens from seventy-eight millimeters on. Vascularization of the lower end of the fibula was noted at sixty-eight millimeters and was present here in all specimens from seventy-eight millimeters on.

In specimens from eighty-five to 125 millimeters, occasional sections of some specimens showed osteoclasts external to the shaft of the tibia, and evidence of removal of substantia compacta. These findings presumably indicate the beginning of reconstruction. Comparable changes were not found in the fibulae of these specimens. Histological evidence of reconstruction (osteoclasts externally, removal of compacta, and exposure of endochondral trabeculae in the metaphysis) became more and more marked in both bones in the older specimens. A systematic study of remodeling was not made. Up to term, a complete *encoche de Ranvier* was not observed.

Skeleton of Foot: Embryonic Period

In some specimens at Stage 17 (eleven to 13.5 millimeters), the tarsus was blastemal and had from three to four digital prolongations. More often, however, a tarsal condensation was first noted at Stage 18 (fourteen to sixteen millimeters). Generally, separate prolongations for the second to fifth toes extended distally. In some specimens at this stage, chondrification had begun. The skeletal elements chondrified in a definite sequence, as pointed out by Senior [56] (for details see O'Rahilly, Gray, and Gardner [71]). All elements except sesamoids were usually chondrifying by Stage 23 (twenty-eight to thirty millimeters).

The second to fourth metatarsals were the first elements in the foot to begin to chondrify (Fig. 3), followed by the cuboid and fifth metatarsal. The navicular was the last tarsal element to begin to chondrify, and the distal phalanx of the fifth toe was the last in the fore part of the foot. Chondrification generally began

TABLE I

INITIAL APPEARANCE OF CARTILAGE CANALS IN THE SKELETAL ELEMENTS OF THE FOOT

Skeletal Element	Length (Millimeters, Crown to Rump) of Smallest Foetus in Which Cartilage Canal Was Found	Length (Millimeters, Crown to Rump) of Smallest Foetus at and after Which Cartilage Canal Was Always Found
Tibia, lower end	60	78
Fibula, lower end	68	78
Talus	43	78
Calcaneus	55	78
Navicular	78	78
Cuboid	83	83
Medial cuneiform	83	95
Intermediate cuneiform	85	107
Lateral cuneiform	85	107
Metatarsal 1		
Base	136	156
Head	104	110
Metatarsal 2		
Base	122	156
Head	136	156
Metatarsal 3		
Base	130	156
Head	156	160
Metatarsal 4		
Base	122	156
Head	156	180
Metatarsal 5		
Base	110	156
Head	156	160
Proximal phalanx 1		
Base	156	160
Head	107	124
Proximal phalanx 2		
Base	156	180
Head	156	180
Proximal phalanx 3		
Base	156	180
Head	156	180
Proximal phalanx 4		
Base	156	180
Head	156	180
Proximal phalanx 5		
Base	144	?
Head	156	?
Middle phalanx 2		
Base	180	235
Head	262	285
Middle phalanx 3		
Base	262	285
Head	262	285
Middle phalanx 4		
Base	262	285
Head	180	285
Center	180	?
Middle phalanx 5		
Base	262	285
Head	180	?
Center	180	?
Distal phalanx 1		
Base	132	180
Distal phalanx 2		
Base	156	262
Distal phalanx 3		
Base	262	?

TABLE I (*Continued*)

Skeletal Element	Length (Millimeters, Crown to Rump) of Smallest Foetus in Which Cartilage Canal Was Found	Length (Millimeters, Crown to Rump) of Smallest Foetus at and after Which Cartilage Canal Was Always Found
Distal phalanx 4		
Base	180	?
Distal phalanx 5		
Base	262	?
Sesamoids		
Interphalangeal of 1	124	?
Metatarsophalangeal		
Medial of 1	85	180
Lateral of 1	118	180

TABLE II

INITIAL APPEARANCE OF BONE IN THE CALCANEUS

Crown-to-Rump Length (*Millimeters*)	Mode of Ossification	Technique	Investigator
93	Periosteal	Histology	Trolle [97]
93	Periosteal	Histology	Present study
95	Periosteal	Ag radiography	O'Rahilly and Meyer [72]
(145 c.-h.)	Periosteal	Schultze	Hasselwander [38]
106	Periosteal	Alizarin red S	Meyer [65]
118	Endochondral	Ag radiography	Meyer [65]
120	?	Alizarin red	Noback and Robertson [67]
122	Parachondral and endochondral	Ag radiography	Meyer [65]
125	Parachondral	Histology	Hintzsche [44]
165	?	Radiography	Fleeker [22]

in the center of each blastemal element. For example, the calcaneus began to chondrify at Stages 18 to 19; the tuber calcanei was not noticeable as a cellular condensation until Stage 20, and did not begin to chondrify until Stage 21. The sustentaculum tali, present as a cellular condensation at Stage 21, was chondrifying by Stage 23. The fifth metatarsal began to chondrify at Stage 18. Its tuberosity, first present as a cellular condensation at Stage 21, was chondrifying by Stage 23.

The digits began to chondrify in proximodistal sequence and, in most specimens at Stage 23, all of their components had begun to chondrify. Hypertrophic cartilage in the shafts of the metatarsals had been noted in earlier stages, and at Stage 23, Phase 3 cartilage was very evident. Less advanced centers of hypertrophy were present in the proximal phalanges. No element of the foot began to ossify during the embryonic period proper. Of thirteen embryos in which all skeletal elements of the foot, except sesamoids, were chondrifying, there was but one in which the middle and distal phalanges of the little toe were fused.

A talocalcanean fusion (talus and posterior part of the sustentaculum tali) was noted in one specimen (Fig. 10), partial fusion between the lateral cuneiform and the third metatarsal in another, and a slight zone of fusion between the calcaneus and the cuboid in another. All fusions were cartilaginous. No accessory cartilages were found, but a bipartite medial cuneiform was observed bilaterally at eighteen millimeters.

PLATE I

Fig. 3: Horizontal section, foot, in a specimen of seventeen millimeters, showing chondrification in the tarsus and metatarsus. The third metatarsal and proximal phalanx are at the right of the lateral cuneiform (arrow). Specimen 17 ROR (× 27.5).

Fig. 4: Frontal section, left foot, in a specimen of twenty-six millimeters, through the fourth metatarsophalangeal joint. Note the condensations for collateral ligaments connected by plantar ligament, deep to which (above) is loose tissue within the joint. Specimen 1439 (× 123).

Fig. 5: Frontal section, left foot, in a specimen of twenty-six millimeters, showing the lateral and medial malleoli, talus, calcaneus, tibialis posterior tendon (right arrow), and peroneus longus tendon (left arrow). Specimen 1439 (× 41).

Fig. 6: Horizontal section, right foot, in a specimen of twenty-eight millimeters. The left arrow indicates the extensor digitorum brevis, the right arrow, the tendon of the tibialis posterior behind the medial malleolus. Note the cavity in the tendon sheath of the tibialis posterior. Specimen 1465 (× 41).

Fig. 7: Horizontal section, right foot, in a specimen of twenty-eight millimeters. The lower part of the illustration shows, from left to right, the calcaneus, talus, and medial malleolus (arrow). Note the fibers of the deltoid ligament from the malleolus to the navicular and, deeply, to the talus. Specimen 1465 (× 41).

Fig. 8: Sagittal section, right foot, in a specimen of twenty-six millimeters. The fibula is at the upper left, then the talus. The calcaneus is at the lower left. Specimen 1439 (× 41).

The various skeletal elements of the foot closely resembled those of the adult in form and arrangement by the end of the embryonic period (Figs. 5 to 9).

Skeleton of Foot: Fetal period

The general configuration of the skeletal elements at various fetal lengths agreed in general with those reported by Hesser [42], Straus [91], Backman [1], and Harris [35].

The talus was the first of the tarsals to show vascular invasion. Cartilage canals were first present at forty-three millimeters, and were constantly present in the talus at and after seventy-eight millimeters (Fig. 34). The vessels within the canals arose mainly from the arteries of the sinus tarsi and tarsal canal. These arteries in the adult are the most important sources of blood supply to the talus [34,102]. Table I shows the initial appearance of canals in the talus and the other tarsals. As in the carpals, the canals increased in number and complexity in older specimens. The number of vascular openings on the surface of each cartilaginous element increased, and anastomoses appeared to develop within the element. Branched, multiple-rooted, and tunnel canals were found.

The calcaneus was the first of the tarsals to begin to ossify (Table II). Periosteal-bone formation, which sometimes preceded endochondral ossification (Figs. 29 and 49), was noted at the inferolateral aspect of the calcanei of at least eight specimens (the smallest was ninety-three millimeters) in which no endochondral ossification was present. Hypertrophied cartilage cells were first noted at 130 millimeters. Endochondral ossification was first noted at 150 millimeters and was constantly present from 180 millimeters on, except in a 302-millimeter specimen in which neither ossification nor cartilage hypertrophy was present. With one exception, in all specimens in which there was endochondral ossification, both endochondral and periosteal bone were present in such a way as to suggest that the endochondral center had been established by the invasion of periosteal buds (Fig. 50). In a 235-millimeter specimen, the endochondral center and periosteal bone were clearly separate. It is thus not possible to state whether the endochondral center consistently begins separately.

Hypertrophied cartilage cells were first noted in the talus at 180 millimeters, cartilage destruction at 235 millimeters, and endochondral ossification at 253 millimeters. With the exception of one 302-millimeter specimen, endochondral ossification was present from 253 millimeters on. In most specimens the endochondral center joined a layer of periosteal bone in the roof of the tarsal canal.

Hypertrophied cartilage cells were first noted in the cuboid at 253 millimeters, and were seen in most specimens thereafter. Endochondral ossification was present at 359 and 373 millimeters. Cartilage hypertrophy was noted in the lateral cuneiform at 348 and 400 millimeters.

Several types of tarsal fusions were noted. Cartilaginous fusions occurred between the body of the talus and the sustentaculum tali in at least three specimens (Fig. 23). The elements were otherwise separate. In one foot a thin, cartilaginous, calcaneonavicular fusion was noted. In the other foot of the same specimen the two elements were connected by a cellular zone. The most common tarsal fusion was a partial one between the lateral cuneiform and the third metatarsal (Fig. 20).

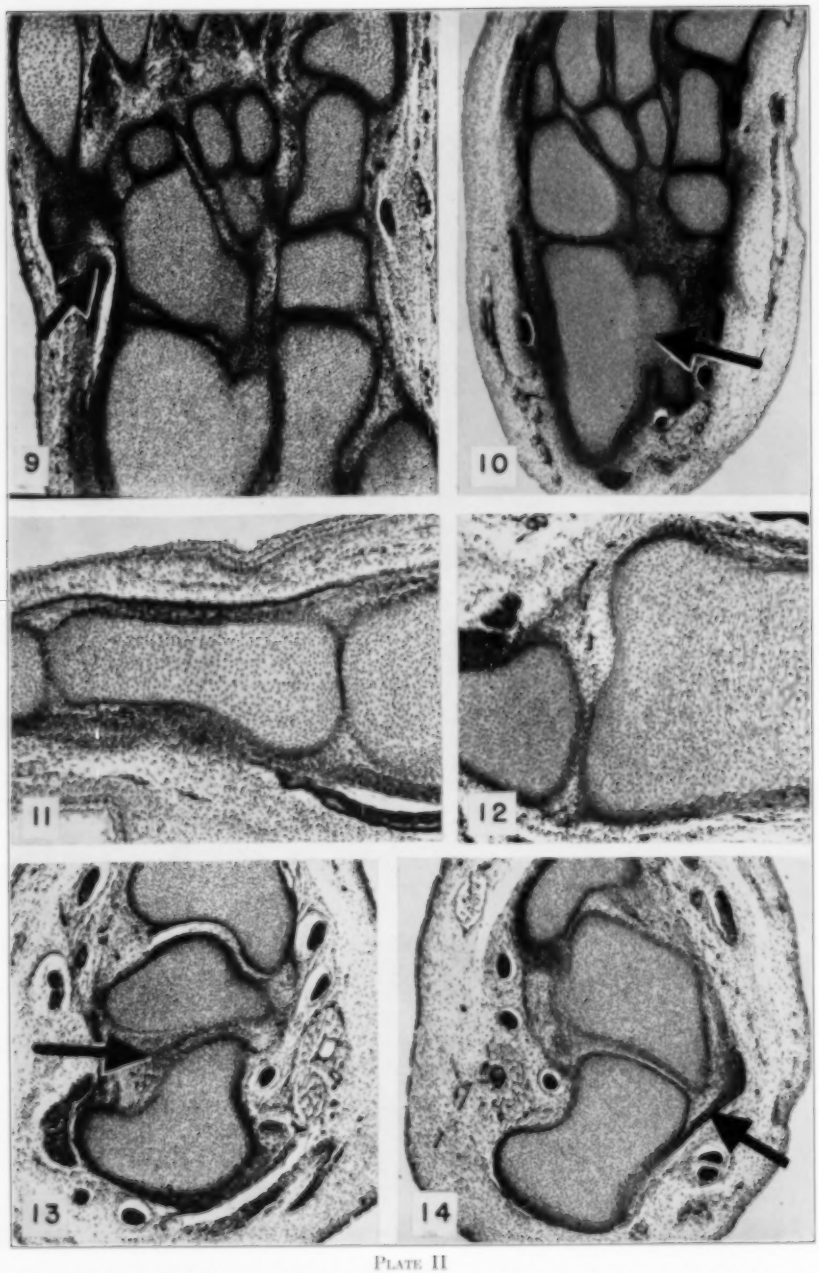

PLATE II

Fig. 9: Horizontal section, right foot, in a specimen of 28.2 millimeters. From below upward: on the left, the calcaneus, the cuboid, and the fourth metatarsal. Note the ligament between the lateral cuneiform and the fourth metatarsal. The peroneus longus tendon (arrow) has a sheath with a cavity. Specimen 28.2 (× 55).

Fig. 10: Horizontal section, right foot, in a specimen of twenty-eight millimeters, showing cartilaginous fusion between the talus (arrow) and the calcaneus. Specimen 1465 (× 41).

Fig. 11: Sagittal section, left foot, in a specimen of thirty millimeters. Note that the fourth proximal phalanx resembles adult element in shape. The head of the fourth metatarsal is at the right. Specimen 906 (× 82).

Fig. 12: Sagittal section, right foot, in a specimen of thirty millimeters. The first metatarsal is at the right and the medial cuneiform is at the left. Note the loose tissue in the joint and capsule, which is penetrated by a few blood vessels. Specimen 906 (× 82).

Fig. 13: Frontal section, foot, in a specimen of thirty-five millimeters. From above downward: tibia, cavity of ankle joint, talus, and calcaneus. The arrow is in the sinus tarsi. Specimen 1503 (× 41).

Fig. 14: Frontal section, foot, in a specimen of thirty-five millimeters. From above downward: medial malleolus, talus, and calcaneus. The arrow indicates the calcaneofibular part of the lateral ligament of the ankle joint. Specimen 1503 (× 41).

Accessory ossicles were noted occasionally; these included separate cartilages in the tendon of the tibialis posterior, an os paracuneiforme (Fig. 37), and an os intermetatarsale. A detailed search for accessory elements was not made.

Intramembranous ossification began at the tips of the distal phalanges during the early part of the fetal period (Table III). Bone at the tip was constantly present from between fifty-five to sixty-five millimeters on (Table III). Vascular invasion of the "membrane bone" was first noted in specimens from sixty to 106 millimeters (Fig. 30), but the constancy of its presence varied greatly (Table III). Vascular invasion of the distal part of the cartilage began between eighty-three and 156 millimeters (Fig. 52). Its constancy likewise varied greatly.

With regard to the other phalanges and to the metatarsals, two phases in bone formation were recorded: the first appearance of a bone collar around the cartilaginous shaft and the first invasion of the cartilaginous shaft by a periosteal bud (Figs. 21, 26, 31, and 45 through 48). The latter was used as a criterion for the approaching onset of endochondral ossification; this, however, does not necessarily begin immediately. In point of fact, an "endochondral center" is not evident until some time after the cartilage is invaded. The initial appearance of a bone collar and of vascular invasion is given in Table III. This table also includes data on ossification from Noback and Robertson [67] and from O'Rahilly and Meyer [72], who employed different techniques. The middle phalanges were the most variable; in some middle phalanges of some specimens there was no bone collar even at term.

During the fetal period, extensive marrow cavities with relatively few endochondral trabeculae were formed in the shafts of the metatarsals and most of the phalanges. No histological evidence of reconstruction was found. By term, characteristic differences were noted in the thickness and regular arrangement of endochondral growth zones, probably reflecting differences in contribution to growth in length. The distal zones of the second to fifth metatarsals were thicker than the proximal ones, whereas the proximal zone of the first metatarsal was thicker. The proximal zones of the proximal and some of the middle phalanges were thicker than the distal zones. Frequently, however, differences in endochondral zones were not as marked as in the hand [27], and in some instances no differences were observed.

Either partial or complete fusion of the middle and distal phalanges of the little toe was observed in about half of the specimens from forty-nine millimeters on (Figs. 28 and 39 through 44). Of eighteen specimens before forty-nine millimeters, only one instance of fusion in the embryonic period and three in the fetal were found.

Vascular invasion of the ends of the metatarsals and phalanges began during the fetal period (Table I). In some specimens, a cartilage canal was noted in the center of a middle phalanx before any bone was present.

Sesamoids

Blastemal condensations for the lateral and medial sesamoids at the metatarsophalangeal joint of the big toe were observed early in the fetal period. They had begun to chondrify in one specimen at thirty millimeters and were almost constantly present in cartilaginous form after forty-three millimeters (Fig. 26). In one foot of one specimen (122 millimeters), only a lateral sesamoid was present. Cartilage canals were first noted in the medial sesamoid at eighty-five millimeters

TABLE III

INITIAL APPEARANCE OF BONE IN THE METATARSALS AND PHALANGES

Skeletal Element	Length (Millimeters, Crown to Rump) of Smallest Foetus in Which Bone Was Found				Length (Millimeters, Crown to Rump) of Smallest Foetus at and after Which Bone Was Always Found			
	Noback and Robertson	O'Rahilly and Meyer*	Present Investigation		Noback and Robertson	O'Rahilly and Meyer*	Present Investigation	
			Bone Collar	Vascular Invasion			Bone Collar	Vascular Invasion
MT1	49	54	43	65	60	62	55	78
MT2	38	(49)	39	60	49	52	55	69
MT3	38	52	39	60	49	52	55	69
MT4	40	52	39	65	51	54	55	69
MT5	45	54	43	65	60	62	55	78
PP1	69	62	69	85	78	96	69	106
PP2	69	62	49	85	76	104	83	107
PP3	88	88	49	85	91	112	95	122
PP4	91	91	85	85	91	114	95	156
PP5	91	91	85	122	115	125	95	156
MP2	116	117	122	156	147		156	180
MP3	140	132	150	235	after 235		308	308
MP4		132	150	262	after 235		359	359
MP5			270	373	after 235		?	?
				at tip / of cart.				at tip / of cart.
DP1	38	(49)	30	60 / 83	49	67	55	78 / 100
DP2	45	54	43	60 / 107	56	126	60	136 / 180
DP3	45	59	43	60 / 125	56	126	55	156 / 180
DP4	45	59	43	69 / 107	68	145	308	308 / 308
DP5	60	62	43	106 / 156	147		308	308 / 308

*In the case of the silverized foetuses, figures from a series (176 foetuses) larger than that previously published are used in Table III. The figure 49 is placed in parentheses because, in the radiographic study, this was the smallest crown-to-rump length employed; it therefore does not necessarily indicate initial appearance.

and in the lateral at 118 millimeters; they were constantly present from 132 millimeters on. The sesamoids were embedded in the fibrocartilaginous-like plantar ligament (Fig. 53), and muscle fibers were attached to them in the same manner as in the adult. In one 100-millimeter specimen two additional sesamoids were found in folds deep to the usual sesamoids (Fig. 27).

An interphalangeal sesamoid of the big toe was first found at fifty millimeters. A sesamoid in this location was present in slightly more than one third of specimens older than this. Each interphalangeal sesamoid presented facets for both the proximal and the distal phalanx. Cartilage canals in such sesamoids (Fig. 54) were first noted at 124 millimeters. In at least one specimen, three interphalangeal sesamoids were noted in a big toe: one was median, and the others were medial and lateral. Metatarsophalangeal (three instances) and interphalangeal sesamoids (two instances at the proximal joint) were rarely present in the little toe (Fig. 39). One instance of a metatarsophalangeal sesamoid in the second toe was recorded.

Interzones: Embryonic Period

Wide, densely cellular interzones were present in metatarsophalangeal joints by Stage 20. In some specimens, there was loosening at the periphery of these areas, deep to the collateral and plantar ligaments (Fig. 4). Also at Stage 20, a densely cellular zone separated the fibula from the calcaneus.

At Stage 21, there were homogeneous interzones in the ankle and in most tarsal joints. In some specimens there was slight loosening at the periphery of the ankle joint. Loose tissue was present at the periphery of the metatarsophalangeal and proximal interphalangeal joints, but the interzones of the distal interphalangeal joints were homogeneous. The interzone separating the fibula from the calcaneus was continuous with that separating the fibula from the talus.

At Stage 22, most interzones were still homogeneous. Loosening at the periphery of the ankle and metatarsophalangeal joints was perhaps a little more noticeable. Likewise at Stage 23 (the end of the embryonic period), most interzones were still homogeneous. Three-layered interzones were restricted mainly to the ankle and the metatarsophalangeal joints. The fibula and calcaneus were still close together but were separated by a part of the talus. Cavitation was beginning in a few joints, as recorded in Figure 1. By the end of the embryonic period, blood vessels had begun to enter the loose tissue adjacent to some of the interzones, particularly in the ankle joint.

Interzones: Fetal Period

Three-layered interzones did not appear in many of the joints until the fetal period (Figs. 11 and 12). The initial appearance of three-layered interzones, like that of cavitation, was variable. A typical interzone did not develop between the lower ends of the tibia and the fibula.

Cavities

Cavitation began in most joints of the foot between approximately the ninth and the eleventh menstrual week. Cavities first appeared generally at the periphery of the joints. The initial appearance of each, in terms of crown-to-rump length, is recorded in Figure 1. Cavities in all joints were not constantly present until after fifty to sixty-five millimeters (Fig. 2). Even at this time, a few were lacking, and many were incomplete in the sense that there were peripheral strands. Many contacts between the various cartilaginous skeletal elements were seen after cavitation had begun, but these were artefacts.

When the lateral cuneiform and third metatarsal were fused, the fusion was often not complete, the region not fused being occupied by either a cellular interzone or a cavity. Likewise, when the middle and distal phalanges of the fifth toe were partially fused, the region not fused was occupied by cellular tissue or a joint cavity. Even when these phalanges were completely fused, there was gener-

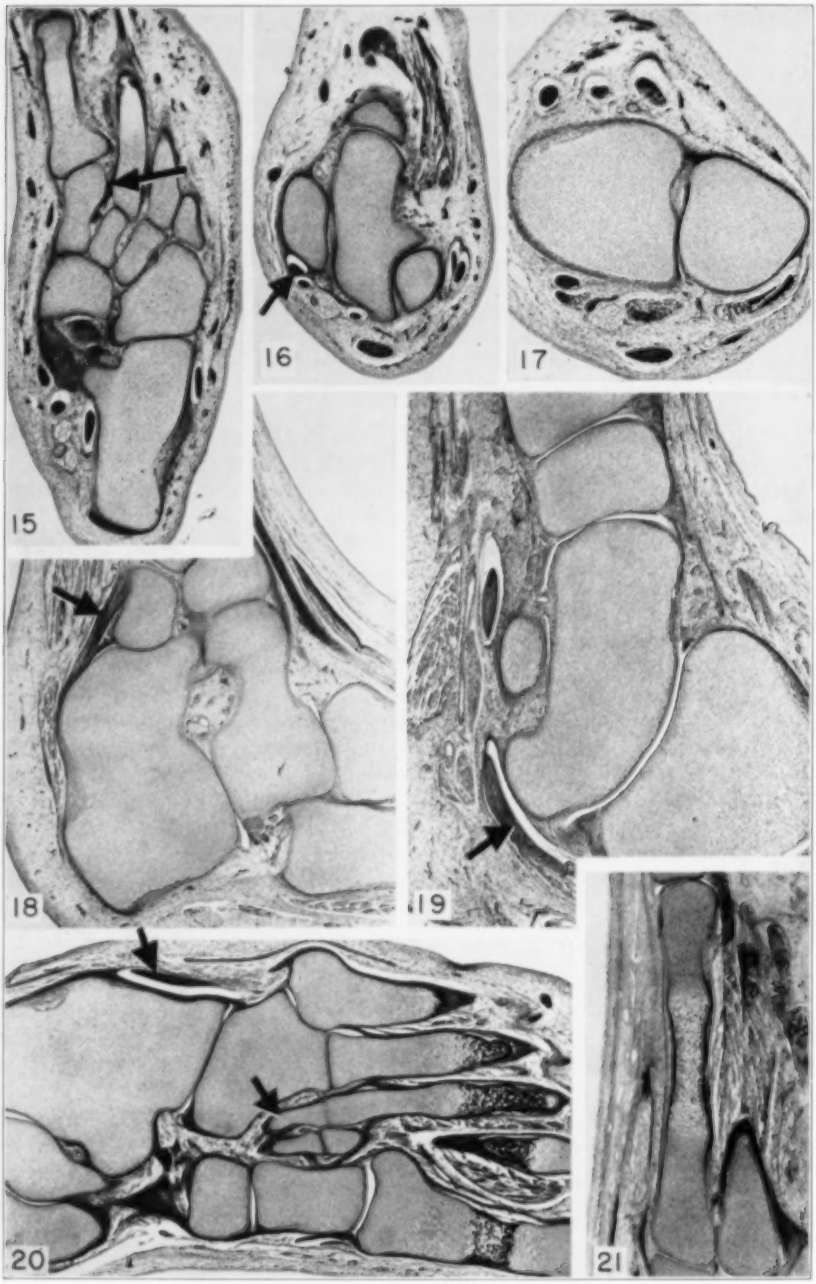

PLATE III

Fig. 15: Horizontal section, left foot, in a specimen of forty-five millimeters, showing the general arrangement. The arrow indicates Lisfranc's ligament (from the medial cuneiform to the second metatarsal). Specimen 1531 (× 18.3).

Fig. 16: Horizontal section, left foot, in a specimen of forty-five millimeters, showing the general arrangement. From left to right: medial malleolus, talus, and calcaneus. The arrow indicates the tibialis posterior tendon. Note the talonavicular cavity above. Specimen 1531 (× 18.3).

Fig. 17: Horizontal section, left foot, in a specimen of forty-five millimeters, showing the

tibia at the left and the fibula at the right, with a small joint cavity behind (below). Specimen 1531 (× 27.5).

Fig. 18: Sagittal section, right foot, in a specimen of sixty millimeters, oriented so that the toes point upward, showing the calcaneus at the left, with the tarsal canal between it and the talus. The arrow indicates the long plantar ligament. Specimen 1024 (× 18.3).

Fig. 19: Sagittal section, right foot, in a specimen of sixty-eight millimeters, oriented so that the toes point upward. The lower part of the illustration shows, from left to right, the sustentaculum tali, talus, and tibia. The arrow indicates the tendon of the flexor hallucis longus; that of the flexor digitorum longus is above it. Specimen 68 ROR (× 18.3).

Fig. 20: Horizontal section, right foot, in a specimen of fifty-five millimeters, showing the calcaneus, upper left, with the peroneus longus tendon and cavity of its sheath above it (upper arrow). The lower arrow indicates the lateral cuneiform, which is fused with the third metatarsal. A zone of slightly increased cellularity marks the partial fusion. Note the calcification of hypertrophied cartilage in the metatarsals. Specimen 44 ROR (× 18.3).

Fig. 21: Sagittal section, left foot, in a specimen of sixty-nine millimeters. The third metatarsal has a bone collar which is beginning to be eroded. The area of erosion (in middle of collar on right), however, is hardly visible at this magnification. Specimen 1122 (× 18.3).

ally a constriction, usually on the lateral side, at the line of fusion. The area of constriction was often filled with vascular tissue.

A cavity appeared between the lower ends of the tibia and fibula by forty-three millimeters and was constantly present thereafter (Fig. 17). It was separate from the ankle joint and did not communicate with the cavity of that joint until sixty-nine millimeters, and not until later did it constantly communicate. A syndesmosis was not observed.

Synovial Tissue

Capillaries had begun to appear in the loose tissue at the periphery of certain joints (especially the ankle) by the end of the embryonic period (Fig. 12), and were noted in the tissue adjacent to initial cavities. In that sense, a synovial membrane could be said to be present. But a definitive membrane with a smooth surface was not present until cavitation was well advanced (by sixty millimeters in most joints, fifty-five in some).

From fifty millimeters on, vessels were very numerous in the areas of future fat pads and synovial folds. Folds were present at sixty millimeters, especially in the ankle, and were thereafter increasingly prominent (Figs. 35 and 36). Villi began to form and were fairly numerous from eighty-three to eighty-five millimeters on. Special nerve stains (silver) were used in only a few specimens. At fifty-eight millimeters, nerve fibers were observed to accompany blood vessels in synovial tissue. Fat was beginning to develop at 106 millimeters. Early fat lobules were observed in some joints at 122 millimeters and were widely distributed from then on.

Fibrous Capsules

Fibrous capsules distinct from ligaments were incomplete or lacking in most joints during the embryonic period. Cellular capsules were noted in some joints, especially in the metatarsophalangeal and interphalangeal joints.

Ligaments: Embryonic Period

Tendons were differentiated by Stage 20, and condensations were observed for the collateral and plantar ligaments of the metatarsophalangeal joints. By the end of the embryonic period, most of the ligaments of the foot and ankle were differentiated as cellular condensations.

Special attention was paid to the ligaments of the sinus tarsi and tarsal canal [89]. In any one specimen it was difficult to identify all the ligaments of this region, because the ease with which they could be distinguished depended on the plane of section. Condensations were observed for the ligamentum cervicis and for another ligament (presumably that of the tarsal canal). The ligamentous arrangement of this region was studied more extensively in the fetal specimens (Figs. 13, 24, and 25).

PLATE IV

Fig. 22: Horizontal section, right foot, in a specimen of sixty-nine millimeters. The arrow indicates the distal prolongation of the tibialis posterior insertion. The upper part of the illustration shows, from left to right, the calcaneus and cuboid and fourth and fifth metatarsals. Specimen 1122 (× 18.3).

Fig. 23: Frontal section, right foot, in a specimen of seventy-three millimeters, showing the tibia and fibula above, the talus and calcaneus below, and talocalcanean fusion in the region of the sustentaculum tali. Specimen 1190 (× 18.3).

Fig. 24: Horizontal section, right foot, in a specimen of eighty-three millimeters, showing the talus (right), navicular (above), and calcaneus (left). Note the cartilage canal in the talus. Section passes through the region of the sinus tarsi. The upper arrow indicates the calca-

neonavicular part of bifurcate ligament, and the lower arrow, the ligament of the canal, at the left end of which a small part of ligamentum cervicis is diverging. See Fig. 25. Specimen 1559 (× 18.3).

Fig. 25: Horizontal section, right foot, in a specimen of eighty-three millimeters, near that of Fig. 24. Arrow indicates the ligamentum cervicis, at the left end of which the ligament of the canal is diverging. Specimen 1559 (× 18.3).

Fig. 26: Sagittal section, left foot, in a specimen of 100 millimeters. From below up: first metatarsal and medial sesamoid, proximal phalanx, and the distal phalanx. See Fig. 27. Specimen 1125 (× 18.3).

Fig. 27: Sagittal section, left foot, in a specimen of 100 millimeters. An accessory sesamoid (arrow) is deep and slightly lateral to the medial sesamoid of Fig. 26. Note the endochondral ossification in the metatarsals, whereas only periosteal bone shows in the phalanx of Fig. 26. Specimen 1125 (× 18.3).

Fig. 28: Sagittal section, left foot, in a specimen of 100 millimeters. The upper element in the illustration is the fused middle and distal phalanges of the little toe. The arrow indicates a small area of bone at the tip of the distal phalanx. Specimen 1125 (× 27.5).

Ligaments: Fetal Period

Smith [89] described the extensor retinaculum and the ligamentous arrangement in the sinus tarsi and tarsal canal in the adult and presented photomicrographs of some of these structures in the foetus. The superficial and deep parts of the sling generally divide into lateral, intermediate, and medial roots. The last is connected to or closely associated with the ligamentum cervicis [50] and the ligament of the canal. A faint condensation for the extensor retinaculum was noted in the present study at twenty-six millimeters. By forty-five millimeters this and other retinacula were becoming thick and cellular. This thickening was characteristically present during the fetal period. By forty-five millimeters the arrangement of ligaments described by Smith was clearly evident and, with minor variations, was present from then on (Figs. 24 and 25).

From thirty millimeters on, all other ligaments of the foot and ankle were present and became increasingly prominent by virtue of their collagenous fibers (Figs. 14, 15, 18, 33, and 55). Variations occurred but were not systematically recorded. From fifty-eight millimeters on, two parts of the plantar calcaneonavicular or spring ligament were noted—one medial and fibrocartilaginous in appearance, the other lateral and fibrous.

A condensation for the deep transverse metatarsal ligament was noted at sixty millimeters. It varied thereafter but was prominent at ninety-five millimeters. The arrangement of this ligament and of the sesamoid mechanism of the big toe was essentially that described for the adult by Haines and McDougall [33].

Bursae and Tendons

Cavitation in the synovial sheath of the peroneus longus and other tendons commonly began during the embryonic period proper (Figs. 7, 9, and 16). At thirty-five millimeters loose tissue was noted in the region of the bursa tendinis calcanei. This bursa was seen at forty-three millimeters, and was generally present from forty-nine millimeters on (Fig. 38). A detailed study of bursae and synovial sheaths was not made.

A systematic study of tendons was not undertaken. The tendon of the tibialis posterior was differentiated by Stage 20, and from then on was very cellular at its insertion into the navicular and at its area of fusion with the spring ligament (Fig. 32). In fetal specimens, it commonly had a fibrocartilaginous appearance here, whereas its distal prolongations were fibrous (Fig. 22). Likewise in the embryo, the tendon of the peroneus longus was cellular opposite the tuberosity of the cuboid; later in the fetal period it became fibrocartilaginous in appearance here (Fig. 51). During the embryonic and the early part of the fetal period, its insertion was mainly into the first metatarsal.

DISCUSSION

The fact that the initial formation of the segments of a limb takes place in

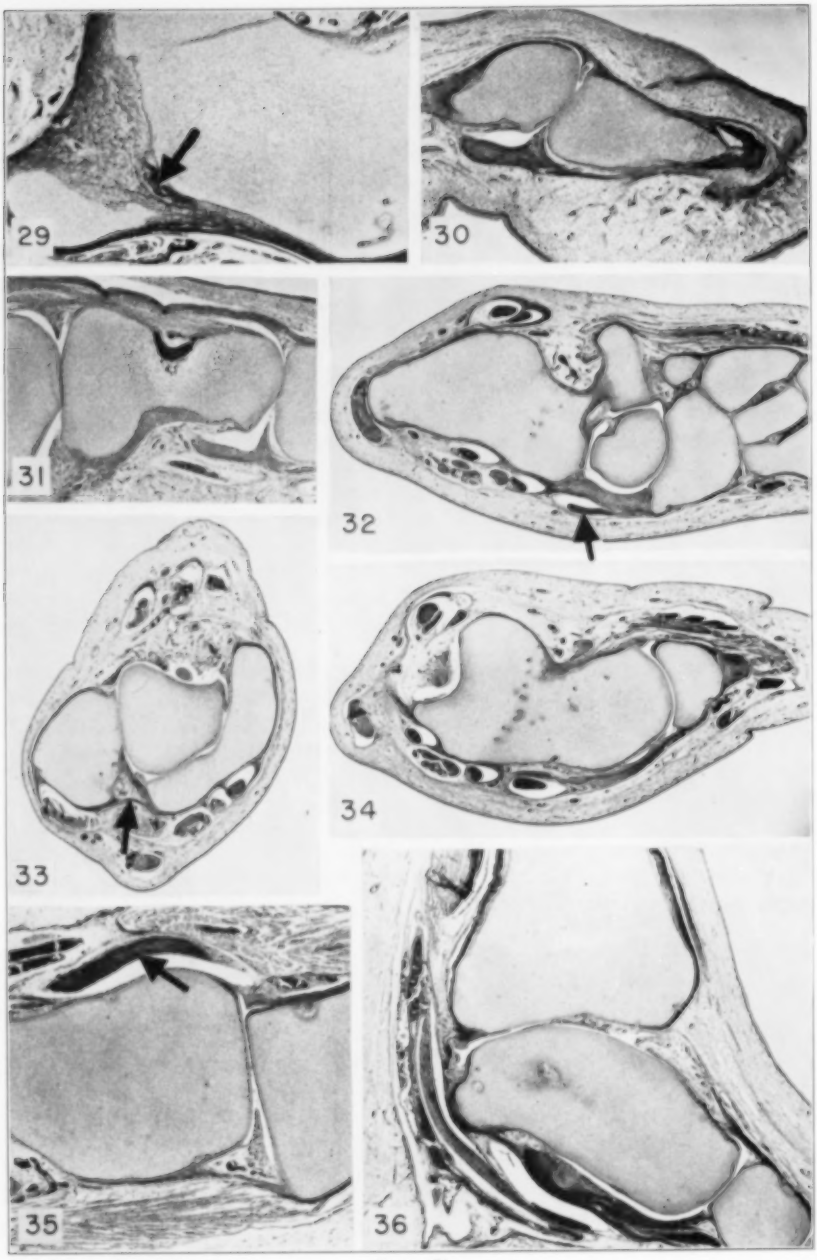

PLATE V

Fig. 29: Sagittal section, left foot, in a specimen of 100 millimeters, showing periosteal bone (arrow) at the postero-inferolateral aspect of the calcaneus. Specimen 1125 (× 18.3).

Fig. 30: Sagittal section, left foot, in a specimen of 100 millimeters, showing intramembranous ossification, with invasion of bone at the tip of the distal phalanx of the big toe. Specimen 1125 (× 18.3).

Fig. 31: Sagittal section, left foot, in a specimen of 100 millimeters, showing eccentric periosteal bone at the proximal phalanx of the big toe. Some calcified cartilage is adjacent. Specimen 100 ROR (× 18.3).

Fig. 32: Horizontal section, right foot, in a specimen of 100 millimeters. The arrow indicates the tibialis posterior tendon, which joins the spring ligament, above which is part of the head of the talus. Specimen 1125 (× 6.6).

Fig. 33: Horizontal section, right foot, in a specimen of 100 millimeters, showing, from left to right, lateral malleolus, talus, medial malleolus, and two tibiotalar ligaments; the more posterior one is indicated by the arrow. Specimen 1125 (× 6.6).

Fig. 34: Horizontal section, right foot, in a specimen of 100 millimeters, showing the talus and navicular. A row of cartilage canals across the talus indicates the entrance of the vessels from the tarsal canal below. Upper right: the extensor digitorum brevis and its tendons. Specimen 1125 (× 6.6).

Fig. 35: Sagittal section, left foot, in a specimen of 100 millimeters, showing the calcaneus at the right and the cuboid at the left, with a vascular, synovial fold in the joint. The arrow indicates the peroneus longus. Only a thin strand separates the cavity of its sheath from that of the calcaneocuboid joint. Specimen 1125 (× 18.3).

Fig. 36: Sagittal section, left foot, in a specimen of 100 millimeters, showing vascular folds in the ankle joint, and the capsule deficient anteriorly. A minute portion of the sustentaculum tali shows below the talus. Specimen 1125 (× 18.3).

proximodistal sequence and in the spatial pattern of subsequent differentiation has been shown by Saunders in experiments on the wings of chick embryos [81]. Morphological studies indicate that the sequential origin of limb parts in proximodistal order is a widespread phenomenon in vertebrates. In the case of the human embryo, the initial formation of the skeletal basis of the limb segments is, in general, proximodistal. Thus, the skeletal elements of the thigh and leg appear as condensed mesenchyme before those of the foot. The femur begins to chondrify before the tibia, and the tibia before the elements of the foot [71]. In the foot, chondrification begins in the metatarsus slightly ahead of the tarsus, but the three rows of phalanges chondrify proximodistally and later than the tarsus and metatarsus. It is of interest to record that, in regard to the total number of skeletal elements that have commenced to chondrify by any given time, the foot is slightly, but distinctly, behind the hand.

It should be emphasized that the phases of blastemal condensation, initial chondrification, and preliminary joint formation occur within the embryonic period proper, and that by the end of the embryonic period (seven weeks after ovulation) the foot resembles that of the adult in most details. It should be emphasized that the generalized blastemal condensation differentiated directly into structures characteristic of man. Many investigators have claimed that, during the ontogeny of the limbs, temporary structures of phylogenetic significance are found. In the case of the human foot, some have reported that structures characteristic of lower vertebrates, such as a prehallux, are present during development [97]. We have found nothing to substantiate such claims.

Differentiation occurs rapidly and in a precise sequence. The differentiation of the blastema into elements arranged like those of the adult occurs from approximately five and a half to seven weeks after ovulation. With the common exception of the distal phalanx of the little toe, all the "canonical" elements of the foot have begun to chondrify by seven weeks after ovulation. The number and arrangement of these elements, therefore, is determined prior to this time. Therefore, anomalies in which the number of skeletal elements is increased arise very early in intra-uterine life, and thus the causative factors must act before the end of the embryonic period. It is possible that, owing to failure of a skeletal element to continue in development, a decrease in the number of skeletal elements may arise after, as well as during, the embryonic period proper. In a study of developmental abnormalities induced in mice by x-rays [80] or by nitrogen mustard [19], it has been found that the critical period during which anomalies of the feet are produced coincides with a very early phase in the development of the limbs. In terms of human embryology, this phase probably corresponds to four to five weeks after ovulation [73]. It is of interest to record that, in the experiments on mice just mentioned, the critical period for the production of overgrowth (for example, polydactylia) was earlier than that for the inducement of reduction (for example, hypodactylia).

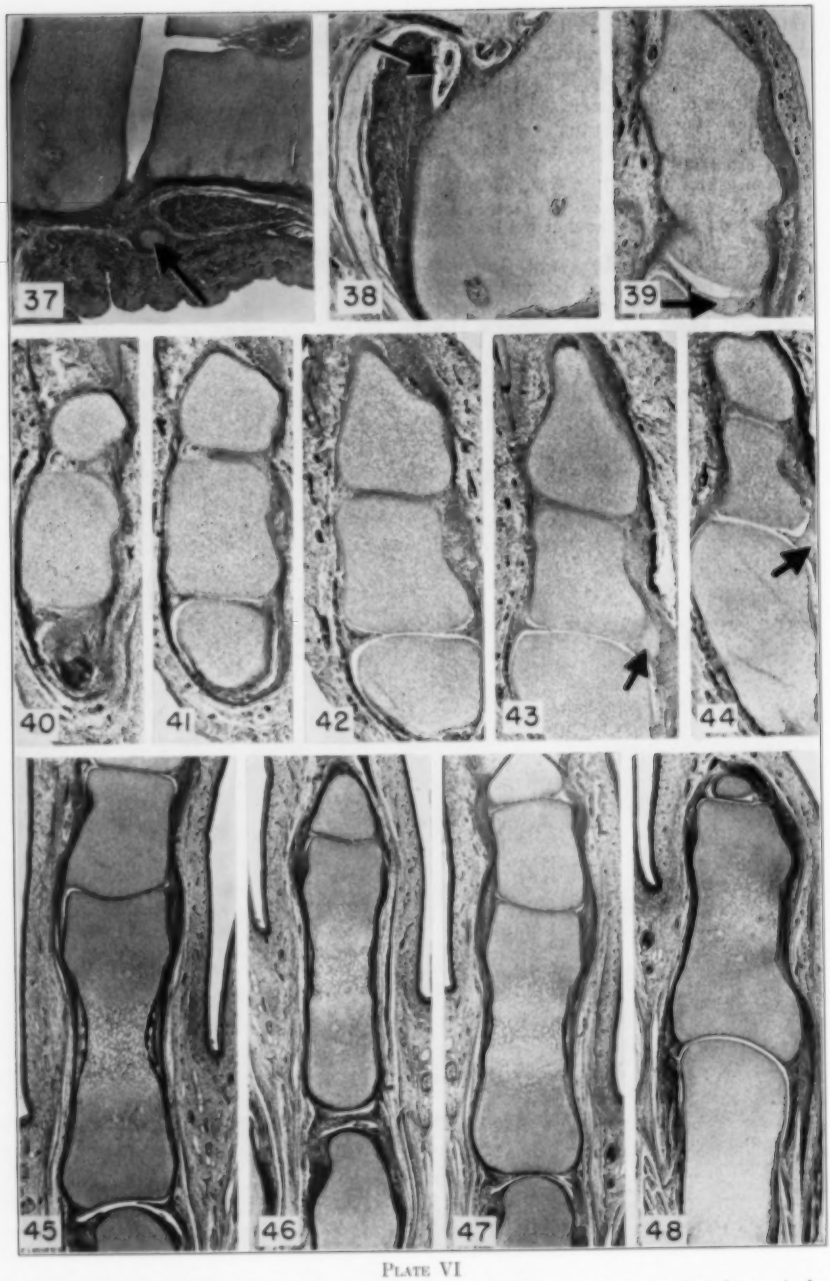

PLATE VI

Fig. 37: Horizontal section, left foot, in a specimen of 122 millimeters, showing the navicular at the left and the medial cuneiform at the right and os paracuneiforme (arrow) near the tibialis anterior tendon. Specimen 122 DBM (× 18.2).

Fig. 38: Horizontal section, right foot, in a specimen of 122 millimeters. Arrow indicates the bursa tendinis calcanei. Note the vascular fold in the bursa. Specimen 122 DBM (× 18.2).

Fig. 39: Completely fused middle and distal phalanges of the fifth toe of left foot, in a specimen of 125 millimeters. Slight constriction and the cellular zone indicate the area of fusion. The arrow indicates a proximal interphalangeal sesamoid (× 27.3).

Figs. 40 through 44 show the middle and distal (upper) phalanges of the right foot of the same specimen (from lateral to medial). Note vascular connective tissue separating them on the lateral side. In going medially, the two fuse, but not completely. On the medial side, a joint cavity separates them. Note the cartilage practically fused with the base of the middle phalanx in Fig. 43 (arrow). In Fig. 44, this cartilage is free as an interphalangeal sesamoid (arrow). Specimen 125 (× 27.3).

Figs. 45 through 48: Horizontal section, left foot, in a specimen of 150 millimeters. From left to right: proximal phalanges of the second, third, fourth, and fifth toes, showing differences in extent of periosteal-bone formation. Note that in the fifth toe, a periosteal collar has not formed around the proximal phalanx. Specimen 1561 (× 18.2).

Joints

The development of the joints of the foot is basically similar to that of the hand [28]. Homogeneous interzones become three-layered, cavities appear in the middle of the three layers, and synovial tissue later lines these cavities. These processes occur also in the shoulder, elbow, hip, and knee joints. The details have been described in recent papers [23,24,26,27]. A comparison of the shoulder, elbow, wrist, hand, hip, knee, ankle, and foot shows that homogeneous interzones generally appear between five and six weeks after ovulation; three-layered interzones are found mostly between five and a half and seven weeks. Cavitation in the larger joints begins usually between six and seven weeks. The smaller joints of the hands and feet may not show a cavity during the embryonic period.

Sesamoids

The sesamoids at the metatarsophalangeal joint of the big toe begin to chondrify shortly after the commencement of the fetal period. The findings on frequency and distribution of cartilaginous sesamoids in this study resemble in general those reported for ossified sesamoids [58]. Thus, sesamoids are found almost always at the metatarsophalangeal joint of the big toe, are found sometimes at the corresponding joint of the little toe, and are found rarely at the other metatarsophalangeal joints. Interphalangeal sesamoids are frequently observed in the big toe and occasionally in the little toe. As in the hand, it seems likely that practically all the sesamoids which appear during fetal life ossify before adulthood. The occasional finding of more than two metatarsophalangeal sesamoids in the big toe indicates that a similar situation in the adult need not necessarily be traumatic in origin.

Cartilage Canals

Cartilage canals appeared during the fetal period in all the skeletal elements of the foot (Table I). Canals were present at both ends of the metatarsals, at the proximal phalanges, and at some of the middle phalanges, although one end of each of these elements is usually ossified from the shaft. Canals were noted first in the head of the first metatarsal and base of the fifth. There is no necessary connection between canal formation and epiphyseal ossification.

In the case of the short bones (tarsals and sesamoids) the canals appear during fetal life, although most of these elements remain unossified until after birth. The appearance of canals is not a reflection of the necessity of nutrition of large blocks of cartilage, as is shown by the early appearance of canals in the sesamoids. The histological appearance of vascularization of cartilage is that of an invasive process.

The order of the first appearance of cartilage canals in the tarsals is not related to either the order of chondrification or to that of ossification. Thus the cuboid is the first to begin to chondrify, the talus is first vascularized, and the calcaneus is the first to begin to ossify.

Ossification

The chief techniques used in the study of the prenatal development of the human skeleton are (1) clearing followed by staining with alizarin red S, (2)

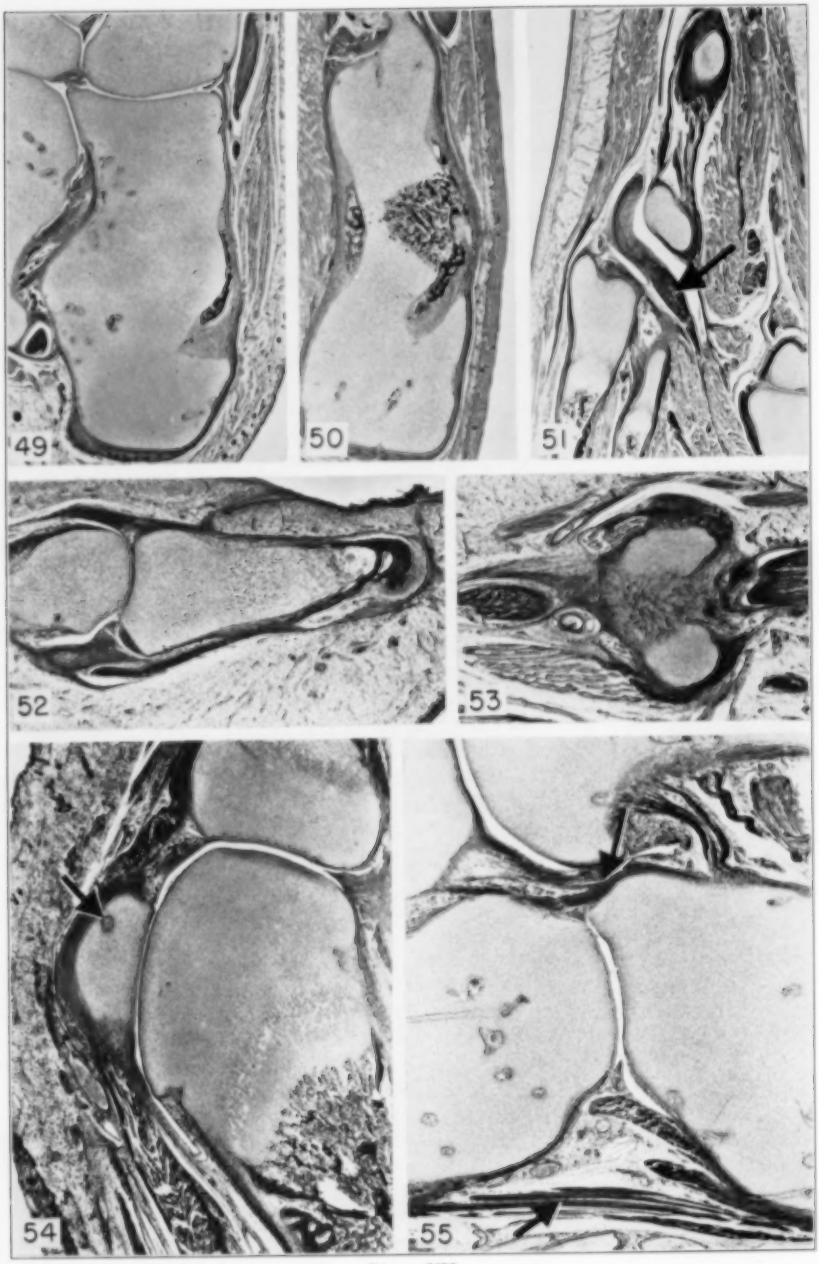

PLATE VII

Fig. 49: Horizontal section, left foot, in a specimen of 124 millimeters, showing periosteal bone on the surface of the calcaneus, at the right side of the illustration. Specimen 124 DBM (× 8.5).

Fig. 50: Horizontal section, left foot, in a specimen of 150 millimeters, showing endochondral ossification extending inward from periosteal bone, on the right side. Note separate periosteal bone at the left side of the illustration. Specimen 150 DBM (× 8.5).

Fig. 51: Horizontal section, left foot, in a specimen of 150 millimeters, showing the peroneus longus tendon turning medially below the cuboid and showing transition from a fibrocartilaginous appearance to a fibrous appearance (arrow), with the fifth metatarsal at the lower left. Specimen 1561 (× 8.5).

Fig. 52: Sagittal section, right foot, in a specimen of 150 millimeters, showing the head of the proximal phalanx of the big toe, with the cartilage canal, at the left, and the distal phalanx with vascular invasion of bone and cartilage. Specimen 1561 (× 18).

Fig. 53: Horizontal section, left foot, in a specimen of 150 millimeters, showing medial and lateral sesamoids of the big toe, joined by a fibrocartilaginous-like plantar ligament. Specimen 1561 (× 18).

Fig. 54: Horizontal section, left foot, in a specimen of 150 millimeters, showing the medial sesamoid of the big toe, with a cartilage canal (arrow); the sesamoid articulates with the first metatarsal. Specimen 1561 (× 18).

Fig. 55: Sagittal section, left foot, in a specimen of 156 millimeters, showing the calcaneus at the right and the cuboid at the left. The lower arrow indicates the long plantar ligament, the upper arrow, the calcanean attachment of the bifurcate ligament; the tarsal canal is at the upper right. Specimen 1551 (× 18).

roentgenography after silver impregnation, and (3) serial sectioning. In a study that involved the employment of three techniques in one specimen [66], it was found that the histological method was the most sensitive and that the first appearance of the alizarin and silver reactions coincided in time with the laying down of the periosteal collars rather than with the onset of endochondral ossification. Only with the advent of the latter event, however, may a "center of ossification" be said to be present. Histological examination is necessary to detect this phase of skeletal development.

A comparison of the figures given in Table III shows that, in the foot, the results of the alizarin and roentgenographic methods in general correspond fairly well with the initial formation of bone collars, although the correspondence is not as close as in the hand.

Whereas ossification commences in the metacarpals and metatarsals at about the same time (bone collars at nine to ten menstrual weeks), ossification in the phalanges of the foot begins approximately three to seven weeks later than in the phalanges of the hand.

The calcaneus is frequently ossified by two processes. An apparently inconstant area of ossification, described as perichondral [39] or parachondral [45], may be seen as early as thirteen menstrual weeks (Table II). It is lateral in position, appearing in a groove between the lateral process of the tuber behind and the peroneal process in front [45]. Thus, it is behind and not, as commonly stated, at the site of the peroneal trochlea or tubercle. It seems likely that a report of bony tissue in the calcaneus of a cleared but unstained foetus of forty-two millimeters [60] was due to mistaking of the opaque cartilage of the calcaneus for bone. As in the case of a long bone (a resemblance stressed by Hasselwander and by Hintzsche), the perichondral or periosteal bone formation is followed by the appearance of an endochondral center [45]. Apparently, two such centers may sometimes appear. At any rate, double centers have been illustrated in infants by several writers (for example, Caffey [12] and Schlüter [82]).

In the present series an endochondral center for the talus was usually present from 253 millimeters on. An ossific center for the talus, however, is not always present at birth [18]. According to one investigator [78], the center for the talus "appears to arise originally from several ossification points which are rapidly united into a uniform center and therefore easily escape observation."

The cuboid sometimes begins to ossify before birth. Its center is said to develop from several foci [39]. The lateral cuneiform rarely presents a center in the new-born [18], and the remaining tarsals commence to ossify during postnatal life.

The distal phalanges: The distal phalanges commence to ossify at about the same time as the metatarsals, that is, early in the fetal period. The proximal and middle phalanges commence to ossify somewhat later. The distal phalanges are peculiar in their mode of ossification. The details have been described by Dixey [20] and Schuscik [85]. The former author pointed out that calcification, intramembranous ossification, and endochondral ossification start at the tip of a distal phalanx instead of at the center of the shaft, as in other "long bones." These processes

then advance in only one direction, namely, proximally. Vessels first invade the bone at the tip and then the cartilage. Not until cartilage at the tip has been removed does endochondral ossification begin.

Accessoria

In addition to the seven "canonical" tarsal elements, a large number of named, accessory skeletal elements have been described (for the relevant literature see O'Rahilly [68,69]). Some of these have been observed (for example by Trolle [97] and Harris [35]) in a cartilaginous state in embryos and foetuses, and some may develop independent ossific centers in postnatal life. Examples of accessoria were encountered during the present investigation, but no attempt was made to assess their incidence. It is generally agreed that the incidence of accessoria in the adult is considerably higher in the tarsus than in the carpus.

Multipartition

The best known example of bipartition in the tarsus is that of the medial cuneiform [3]. Prenatal instances of this condition have been recorded (for example by Trolle [97]) and one was observed in an embryo of the present series.

Fusion

The term *fusion* is used in the hand and foot for the occurrence as a single structure of something which is usually composed of two or more elements. Examples in the foot include talocalcanean and calcaneonavicular fusions, both of which conditions may be of clinical importance [14,36,37,41,87,88,100,101]. Prenatal instances of both of these conditions have been recorded (for example, by Trolle [97]) and were also seen in the present study. The embryological relationship of skeletal fusions to cavitation needs further investigation.

Symphalangia, or so-called fusion between the phalanges, is found frequently in the little toe between the middle and distal phalanges. Venning [98,99] reports the incidence in adults as about 43 per cent, and Pesonen [74] as 29 to 34 per cent. The incidence among Japanese, however, is 72 per cent or more [74,97]. In foetuses older than twelve weeks, an incidence of 36 per cent has been reported [97]. In the present series, either partial or complete fusion of these phalanges was noted in about half the specimens from forty-nine millimeters on, whereas it was observed in only three specimens smaller than forty-nine millimeters—one in the embryonic and two in the fetal period. The incidence was somewhat higher than that reported by Trolle [97].

It is a distinct possibility that middle and distal phalanges which are going to fuse chondrify separately and then undergo cartilaginous fusion. Hence the incidence rises after the fetal period begins. The fusion is not always complete; in some specimens the two elements show cartilaginous fusion on one side, with connective tissue or a joint cavity intervening on the other. The incidence of tarsal and phalangeal fusions might well be higher in the foetus than in the adult. Some are partial and tenuous and could easily break down when weight-bearing begins.

SUMMARY

One hundred and eighty-four human embryos and foetuses were studied. They ranged from seven to 460 millimeters in length.

The skeleton of the foot was blastemal by Stage 17, and the various elements began to chondrify between Stages 18 and 23, that is, from approximately five and a half to seven weeks after ovulation. Chondrification occurred in a definite sequence.

Cartilage canals were first seen at forty-three millimeters; they were found in all skeletal elements, including sesamoids. Their first appearance occurred in a definite sequence.

Ossification was first noted in the metatarsals and distal phalanges early in the fetal period, and later in the proximal and middle phalanges. Some middle

phalanges had not begun to ossify at term. Periosteal ossification was first noted in the calcaneus at ninety-three millimeters, and endochondral ossification at 150 millimeters (about five months). Endochondral ossification was first noted in the talus at 253 millimeters (about eight months). In two specimens at term, endochondral ossification was beginning in the cuboid. Cartilaginous sesamoids were first noted at thirty millimeters; their distribution and frequency during the fetal period roughly corresponded to those reported for the adult.

Fusions were noted as follows: talocalcanean, calcaneonavicular, cuneometatarsal, and interphalangeal. Fusion of the middle and distal phalanges of the little toe was common. Accessory ossicles that were noted included os paracuneiforme, os intermetatarsale, and a separate cartilage in the tendon of the tibialis posterior.

Homogeneous interzones were present in the joints of the foot by Stages 20 to 21. Cavitation began in a few joints during the embryonic period, but the formation of three-layered interzones, followed by cavitation, began in most joints during the early fetal period.

Capsules and ligaments were present as cellular condensations before cavities and, in some cases, before three-layered interzones appeared.

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DISCUSSION

Congenital Dislocation of the Hip

(Continued from page 846)

Dr. J. Hiram Kite, Atlanta, Georgia: I hadn't planned to say anything, but I am very much interested in these hips. The interest is this—what is it that causes aseptic necrosis? We have been told that it is because we do not pull the hips down and thus cause trauma to the head in reducing the dislocation. Now if that is true, then we should pull these heads down. However, this entails hospitalization of the infant for four weeks which is a financial strain on the parents. Nevertheless, if we can get better hips, then we must do something about it. I reviewed the hips (including bilateral cases) on my service and we have had just as much aseptic necrosis in the heads we pulled down as in the ones we did not. Our critics say that we did not pull them down well, but our roentgenograms disprove this. Is, then, aseptic necrosis due to the trauma of reduction, or might it be due to some other factor? I would suggest that it is atrophy since at no other time do we hold bone so long in immobilization —six to seven months.

Dr. H. Relton McCarroll, St. Louis, Missouri: Dr. Ponseti says that if you are dealing with a dysplastic hip, or what he calls a preluxation, and if it is not treated, it will progress to a true dislocation. It is possible, of course, for many of these dysplastic hips to straighten out spontaneously, without treatment. Treatment, however, is justified in all instances because it is so mild. The point I want to make is this, that if we do have a persistent dysplastic hip, it does not occur in the form of an anterior or posterior dislocation, it simply rides superiorly in the acetabulum, and this is what we have always called a superior or rather an upward subluxation. It is entirely different from the anterior or posterior position of this hip and they are entirely two different problems. I would like to indicate that there are two distinct clinical entities that we are dealing with here, one is a primary dislocation and the other is a primary dysplastic hip. They are entirely different. You can separate them in the infant and their management is entirely different.

Dr. Ponseti (closing): I disagree with Dr. McCarroll that the dysplastic hip or the preluxation is different from the dislocation. We had five cases of preluxation in which the treatment was refused by the parents and dislocation developed. When a positive Ortolani sign is encountered the child must be treated. If he is not treated the hip will dislocate.

In Caffey's paper, published about two years ago, it is indicated that preluxated hips usually develop well without treatment. These do well without treatment if they have a negative Ortolani sign, but when there is a positive Ortolani sign, I am sure that most of them will dislocate.

A point now about the positioning of the hips. I feel sure that the aseptic necrosis of the head of the femur is related to reduction. The necrosis is seen in high dislocations reduced without previous traction or in cases immobilized in the first position with the thighs in too much abduction. In this position the head of the femur is pressed with too great force against the acetabulum; I am also quite sure that in dislocation of the hips in children, the best position after reduction is with the thighs in flexion of 90 degrees (or 100 degrees maximum) and abduction of 70 or 80 degrees, because in this position the head of the femur is well centered in the acetabulum and can build a deeper acetabulum than if the second position is used.

We should, by all means, try to see in our communities these children early, preferably under one year of age, and then the result of treatment will be almost uniformly good. I think it is of great discredit to our profession that we see these children after they have started walking, and certainly if we see them after two years of age. This should happen only in the jungle, but not in the modern United States.

Frozen-Shoulder Syndrome in Patients with Pulmonary Tuberculosis

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As consultant in orthopaedic surgery to several tuberculosis sanitaria, I have long noted the high incidence of the so-called frozen-shoulder syndrome among the patients in these hospitals. Although it is well known that this syndrome is often associated with cardiac and other debilitating illnesses [2,5], the literature is quite sparse concerning its relationship with pulmonary tuberculosis.

Franklin and Nemcik noted a high incidence of shoulder complaints of varying degree in tuberculous patients at Fitzsimons Army Hospital. These patients were not followed for any length of time, but the authors were discouraged by the poor results obtained by any method of treatment. Einaudi recently reported a high incidence of periarthritis of the shoulder in tuberculous patients in Italy, but he too did not have any long-term follow-up of these cases.

In order to clarify not only the incidence but the clinical course and eventual result of this condition, I decided to follow all cases seen in one hospital in a given period of time. Mount Wilson State Hospital was selected—a modern, efficient institution near Baltimore—in which patients of all ages with all degrees of tuberculosis are treated. The calendar year of 1956 was chosen, and all patients with the frozen-shoulder syndrome who were seen by me in that period were included in the series. This obviously meant, in a chronic condition such as this, that some patients first had distress with their shoulders in 1955, others continued to have difficulty on into 1957, but all had been afflicted to some degree in 1956.

The next problem was to clarify what was to be included under the term *frozen shoulder*. Many patients had pain and some degree of stiffness without real "freezing" ever developing. Therefore, an arbitrary minimum of 50 per cent limitation of scapulohumeral motion was set; this was further defined as inability to abduct the shoulder either actively or passively beyond 90 degrees. The grading of percentage of stiffness was necessarily rough because factors like apprehension and pain could not be excluded, but with practice a fairly accurate estimate could be made.

Incidence

Twenty-nine patients with at least 50 per cent limitation of shoulder motion were seen in 1956; seven had bilateral limitation. There was thus a total of thirty-six shoulders. Most remarkable of all, and a tribute to the advantages of study in a tuberculosis sanitarium, all patients were followed for at least six months, or until recovery.

In 1956, the average daily census of the hospital was 408 patients, so the probability of frozen shoulder was 7.1 per cent per patient year. The average hospital stay was 277 days, so only 904 patients were in the hospital during the year, some for as little as a day, some for the entire year. Counting total patients for 1956, the incidence of frozen shoulder was 3.2 per cent.

Age

The patients with frozen shoulders ranged in age from thirty-seven to sixty-eight years; only one was under forty, and only three over sixty years old, with the average being 50.8 years. In the hospital at large the average census was 170

patients in the group of patients forty to sixty years old. As our series showed twenty-five patients in this age group, the average incidence per hospital year in the forty to sixty-year group was 14.7 per cent (nearly one case in six). In the group under forty, the incidence was a minute 0.55 per cent, and in the group over sixty years, only 3.7 per cent.

Sex

In this series there were twenty men and nine women, showing what seemed to be a preponderance of men. However, the hospital averaged 67 per cent men and 33 per cent women, so the final ratio was 7.3 per cent men and 6.6 per cent women.

Side of Body with Shoulder Lesion

It was believed that the side of the body with shoulder stiffness might be of significance. However, in this series there were seven patients affected bilaterally, ten patients with the lesion on the right side, and twelve with the lesion on the left side, giving seventeen (47 per cent) right shoulders and nineteen (53 per cent) left shoulders.

Side of Body with Pulmonary Lesion

There are numerous theories that periarthritis of the shoulder is related to reflex neurological changes stimulated by pathological changes elsewhere. Following this line of thought, it would be logical to expect the pathological changes to predominate in an area with the same general reflex arc as the affected shoulder and it was anticipated that there would be some correlation of the side of the body with the lung lesion with the side of the body with the affected shoulder. Instead, it was found that the major lung lesions were on the same side as the frozen shoulder in only 13 per cent and were on the opposite side in 27 per cent, whereas the lungs were equally affected or both shoulders were involved in 60 per cent of the patients. The apparent predilection for the opposite side was not considered to be statistically significant in this small series, and it was felt that the figures show only that the side of the body with the lung lesion is not important.

Severity of Lung Lesion

There was, however, a definite correlation between frozen shoulders and severity of the pulmonary tuberculosis. In 13 per cent of our patients the tuberculosis was mild; in 35 per cent, moderate; and in 52 per cent, severe.

Duration of Hospitalization

Similarly, there seemed to be a definite connection between the length of hospitalization and the onset of shoulder symptoms. Only 21 per cent of the patients had onset of shoulder symptoms in the first three months of hospitalization, 58 per cent in the three to six-month period, and 21 per cent after six months. It is interesting to note that none of the patients, except two transfers from another sanitarium, had frozen shoulders before admission.

It is the general policy of this and other tuberculosis hospitals to put patients, especially those with severe or moderately severe lesions, on relative bed rest for at least three to six months. Excessive exercise is discouraged, and sometimes overemphasis on rest and dire predictions of the consequences of activity are needed to restrain recalcitrant patients.

All patients were receiving antituberculosis drugs, and many were receiving a combination of drugs. Therefore, an accurate correlation between any one drug and frozen shoulder was impossible.

From the figures, it seems that the debilitated person of from forty to sixty years of age, after three months of bed rest, is the ideal candidate for a frozen shoulder.

Clinical Course

I was eager to follow these patients to see how their clinical course and end results compared with those patients with frozen shoulder seen elsewhere. Fortunately, all of these patients received practically the same treatment for their shoulders. This treatment was essentially *laissez faire* or watchful waiting—just symptomatic medication and occasional application of heat—so that the natural course of the condition could be observed. The primary approach was to reassure the patient and to explain that although the condition might worsen before improving eventual recovery was practically certain. It was even more valuable to refer them to someone on their ward or just down the hall, who had recovered from the same condition.

Physical therapy was not available at first and was not requested thereafter, as the patients seemed to be doing well. Cortisone was decided against for these patients with active tuberculosis. All patients in the active inflammatory phase of periarthritis were advised to use their arms as much as possible in motions that did not hurt, but not to try painful stretching. Patients in the healing phase were advised to do, and shown how to do, wall climbing, rope pulling, and other exercises, but there was little control over how well these exercises were done.

Objective evaluation was primarily concerned with the limitation of motion associated with the frozen-shoulder syndrome. According to my evaluation, in twenty-six shoulders (72 per cent) of those involved there was, at worst, 50 to 65 per cent limitation of glenohumeral motion; in four shoulders (11 per cent) there was 65 to 80 per cent limitation; and in five shoulders (16 per cent) there was over 80 per cent loss of motion.

In the average frozen shoulder there are two simple and distinct clinical phases—one of increasing pain and stiffness, and one of gradually decreasing pain and stiffness. Although the dividing line is not abrupt, most patients are able to recognize, within a few weeks, when the tide turned and improvement started.

In this series, increasing pain and stiffness lasted less than three months in only four patients (14 per cent); over four months in only three patients (10 per cent); and three to four months in twenty-two patients (76 per cent).

The duration of decreasing stiffness was harder to evaluate as some patients would show slight improvement, especially in range of motion, for many months after a good functional recovery had been attained. Eventually, a point was selected at which the patient was no longer significantly bothered by the arm and used it freely. This usually coincided with at least 80 per cent of normal range of motion.

In five patients (17 per cent) the duration of decreasing stiffness to relative recovery was less than three months. In seven patients (24 per cent) it was greater than four months. Again, however, for the majority, seventeen patients (59 per cent), the duration was three to four months.

Thus, the total duration of the condition was under six months in four patients (14 per cent), six to eight months in twenty-one patients (72 per cent), and over eight months in four patients (14 per cent).

The evaluation of the final result is also somewhat inaccurate as some patients were last seen only six to eight months after onset of their symptoms. In these a finding of 80 per cent of normal motion would probably, in a few more months, change to 90 or 100 per cent. A few other patients were followed closely for nine months to a year before they attained satisfactory motion; they were then discharged from further observation.

I classified the final range of motion in sixteen shoulders (44 per cent) to be 95 to 100 per cent of normal; fourteen shoulders (39 per cent) to be 90 to 95 per cent of normal; four (11 per cent) to be 80 per cent; and two shoulders (6 per cent) to be 70 per cent. These last two shoulders were complicated by surgery; one patient had a bronchocutaneous fistula which had completely scarred down his scapula, and one patient had thoracoplasty on the affected side.

Subjective Clinical Course

The second major clinical characteristic of the frozen-shoulder syndrome is the purely subjective one of pain. This is very hard to evaluate accurately but most patients can be classified rather easily as having mild, moderate, or severe pain. Almost all patients will have pain on stretching or strenuous use of the involved arm. Most patients have fairly comfortable use of their arms during light activity, with the elbow near the side. Therefore, pain at rest—particularly at night—was felt to be a more accurate guide of the degree of severity.

All patients were asked about night pain. If they rarely lost more than an hour's sleep because of pain, the pain was classified as mild. If they lost one to three hours' sleep for one to three weeks, pain was considered moderate. Anything worse than this was classified as severe.

When this criterion was used, fifteen cases (52 per cent) were considered mild; five cases (17 per cent), moderate; and nine cases (31 per cent), severe.

Localization of Pain

All patients were asked to localize the areas of greatest pain and tenderness. Almost all said that the pain moved about, sometimes worse on top of the shoulder, sometimes over the lateral part of the shoulder and upper part of the arm, and, occasionally, in the forearm. The most common area was the lateral portion of the upper part of the arm, near the deltoid insertion. This also seemed to be the area of maximum tenderness in many patients, although most, especially those whose pain was in the acute phase, had tenderness in many areas. The bicipital groove was frequently tender but rarely more so than other areas, including the nearby coracoid process. Biceps-tendon tests gave no more positive results than other tests that put strain on an acutely inflamed shoulder.

Psychological Reaction

Most authors have noted a high correlation between nervous and emotionally unstable persons and frozen shoulder. Coventry even went so far as to describe a "periarthritic personality". To evaluate this aspect I examined the patients' charts for a history of neurotic or unstable behavior either before or during hospitalization. In addition, the patients were asked to evaluate their own nervous behavior —whether they were high-strung, tense, or subject to frequent maladies. It was realized that hospitalization for tuberculosis was an unsettling experience, however, all of these patients were hospitalized and many actually were relieved of anxiety by having somebody else look after them.

When both the medical evaluation and the patient's own interpretation were used, fourteen patients (48 per cent) were classified as having stable personalities, seven patients (24 per cent) as being moderately stable, and eight patients (28 per cent) as having basically unstable personalities. I felt that in this series there was no such thing as a periarthritic personality, and that the malady could develop in anyone, whether emotionally stable or unstable.

There was, however, as might be expected, a definite correlation between previous emotional instability and the patients' nervous reactions to the frozen shoulder. Of the eight patients classified as emotionally unstable, five had severe nervous reactions to their shoulder symptoms, and three had moderately severe reactions.

Also, as might be expected, there was considerable correlation between degree of pain and nervous reaction. Of the nine patients whose pain was classified as severe, six had severe nervous reactions; one, moderate; and two, mild. The question always arises whether it is the pain that causes the nervous reaction or the psychic element that increases the apparent pain. This is, of course, impossible to answer, but it can be noted that of those patients complaining of severe pain, only two had severe objective stiffness; three, moderate stiffness; and four, mild stiffness.

It is my opinion that even though the incidence and objective symptoms of the frozen-shoulder syndrome are not directly connected to the patient's temperament, the subjective symptoms are. Since nervous people, complaining of great pain, make the greatest noise and fuss, the erroneous impression has been created that they form the great majority of sufferers.

Previous Shoulder Problems

All patients were questioned about previous shoulder problems in an effort to see if these could be initiating or predisposing factors. A remarkable feature that evolved from this study was the relative lack of shoulder difficulty in the months or years preceding the onset of the frozen shoulder. Of twenty-nine cases only one patient had a clear-cut history of bursitis with calcium deposit. One had had a recent wrist fracture with probable simultaneous contusion of the shoulder. Two patients had thoracoplasty; one shortly after the onset of shoulder symptoms, and one when the frozen shoulder seemed to be improving. In both patients the shoulders became considerably worse for several months; one had tuberculous osteomyelitis of the upper metaphysis of the humerus of the involved shoulder.

I was impressed by the lack of any history of chronic disability of the shoulder as well as the lack of recent disease or injury that could have triggered the onset of the frozen shoulder. Some of the patients on whom pulmonary operations were performed during the course of the active inflammation were made worse, just as some patients are made worse by ill advised manipulation or too vigorous physical therapy.

Roentgenograms were made in a number of cases and in general showed only mild osteoporosis and the so-called high shoulder with the humerus riding a little high in the glenoid, and the space between the acromion and the head of the humerus slightly diminished.

Associated Stiffness in Other Joints

Some diffuse stiffness in the fingers developed in six patients, in the same side as the shoulder involvement. In three the stiffness was quite severe; these cases could be classed as typical of the shoulder-hand syndrome. As usual in these cases, the hands limbered up much more slowly and less completely than the shoulders.

Two patients had mild swelling and stiffness of the knees and two had chronic back pain. One man had symptoms of mild generalized rheumatoid arthritis with occasional swollen joints. One patient had bilateral Dupuytren's contracture and one, a trigger-thumb. In general, however, the frozen shoulder seemed to be a local condition rather than part of a systemic rheumatic reaction.

Other Diseases

All of the patients with frozen shoulder obviously had pulmonary tuberculosis of greater or lesser degree. Several had pulmonary complications such as emphysema or empyema. A few had minor cardiac conditions. In general, however, there were few other complicating diseases and no evidence that any influenced the shoulder condition, except in those few patients, previously mentioned, who had thoracic surgery and infection in the shoulder-girdle area.

Subfrozen Shoulders

Many patients complained of aching and frequently of some stiffness; however, their shoulders could not be included in this series because 50 per cent limitation of motion was never reached. Also, of the twenty-two patients in this series with only one frozen shoulder recorded as such, six had some degree of pain and stiffness in the opposite shoulder.

It is apparent that not only do we not know the cause of this condition, but

we cannot predict how severe the condition will become or whether it will develop at all.

CONCLUSIONS

This study brings out several convincing statistical conclusions of a positive nature:

1. The high incidence of frozen shoulder in a tuberculosis sanitarium.
2. The great majority of patients were in the age group of forty to sixty years.
3. The onset is generally three to six months after admission to hospital and after prolonged bed rest.
4. The majority of cases occurred in patients with fairly severe lung disease and general debility.
5. The clinical course is fairly long, but relatively mild.
6. The end results are good despite minimal therapy.

There are also several convincing conclusions of a negative nature. There is:

1. No significant variation according to sex.
2. No significant predilection for shoulder lesion according to a particular side of the body.
3. No significant correlation with pulmonary lesion according to a particular side of the body.
4. No significant correlation with previous shoulder symptoms.
5. No significant correlation with patients' nervous reaction or the periarthritic personality.

COMMENT

Periarthritis of the shoulder in the patients, seen by me, is probably due to bed rest and relative disuse in a debilitated person who is at the age when normal degenerative changes are occurring in the shoulder cuff and other structures. There may be an endocrine factor as this is the so-called menopausal period, and the debilitated patient probably has, in addition, adrenocortical insufficiency. Antituberculosis medication could be a factor, but no evidence for this was found.

Preventive treatment, consisting in regular arm exercises for the patients forty to sixty years old who are on bed rest, should be considered.

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Final Report of the Committee for the Study of Femoral-Head Prostheses*

The results of two previous surveys on the use of femoral-head prostheses were reported in 1953 and 1954. In these surveys an attempt was made to ascertain the number of members of The Academy using hip prostheses and the type of prosthesis being employed. An evaluation of the results was not attempted then since insufficient time had elapsed. This third survey, three years later, was designed to obtain, in addition to other data, statistics on the results of these procedures. In 1957 all members of The Academy received questionnaires; these were four pages long and required a considerable amount of time and effort to fill out. The following information was obtained:

To the question: "Are you now using femoral-head prostheses?", 453 members replied in the affirmative, eighty-eight in the negative, and ten answered incompletely. There were then 551 replies to this question.

The 453 members of The Academy who were using prostheses had performed 10,059 unilateral and 96 bilateral procedures. The eighty-eight members who were not using prostheses had performed 119 procedures before discontinuing the use of prostheses. This made a total of 10,274 procedures.

The patients ranged in age from two to 107 years—an average age of 86.7 years.

The follow-up time in months after operation ranged from one to 120 months. The average follow-up time was 54.35 months.

The number of surgeons using the various types of prostheses were: 195, Moore; 134, Thompson; 76, Eicher; 19, Naden-Reith; 10, Minneapolis; and 4, Judet. There were eighteen other types of prostheses, none of which had been used by more than five men.

Replies to the question: "What type of prosthesis did you formerly use?" indicated that rather striking changes had occurred in the numbers of surgeons using the various prostheses, as shown by the following: 137, Judet; 92, Eicher; 64, Naden-Reith; 30, Thompson; 24, Moore; 21, Judet nylon; 16, Collison; and 8, Minneapolis. There were sixteen other prostheses, each of which had been used by about eight surgeons.

From these data it is apparent that there has been a very definite trend toward the use of an intramedullary-stem prosthesis in preference to the short-stem varieties, and nylon has been abandoned completely.

The reasons given for the change and the number of surgeons giving each of these reasons were: Judet wobbled and became loose, 27; acrylic wore down or broke, 25; intramedullary type is better, with more stability, 19; Moore gives better rotation of the hip, 18; Naden-Reith was unstable and drifted into varus, 13; nylon fractured, 12; Judet stem too short, resulting in varus, 10; Thompson easier to apply, 7; Collison fractured, 6; Eicher too heavy, 3; J. E. M. Thomson drifted into varus as the neck absorbed, 3; and Thomson became loose, 3. There were nineteen other reasons for change.

The indications for the use of femoral-head prostheses and the number of surgeons mentioning these indications were: subcapital fractures in patients over sixty to seventy years of age, 225; aseptic necrosis and fragmentation of the femoral head, 186; non-union of fractures of the femoral neck, 178; hypertrophic arthritis, traumatic arthritis, and malunited fractures, 163; rheumatoid arthritis, 51; path-

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ological fracture or tumors and radiation necrosis, 22; loss of reduction or failure after reduction and fixation with a nail, 19; Legg-Calvé-Perthes disease or old, slipped capital femoral epiphysis, 15; bilateral hip disease, 12; and Parkinson's disease, 6. Twenty other indications were also mentioned.

It is interesting to note that in the preliminary survey the use of a prosthesis in a fresh subcapital fracture was in fourth place. In this survey this indication now becomes the most common.

Similar data on the contra-indications to the use of these prostheses were: infection (including tuberculous), 156; acute fracture in patients under sixty years of age, 110; adolescence, 65; debility or poor surgical risk, 56; osteoporosis, 48; rheumatoid arthritis, 38; osteo-arthritis, 26; cystic acetabular disease, 25; any remaining head, 13; and where cup would be better, 12. Thirty-eight other contra-indications were also recorded.

These figures show that any previous infection is considered a primary contra-indication. Many surgeons believe that fresh fractures of the hip should not be treated with a prosthesis if the patient is under sixty years of age.

The value of a prosthesis in treating rheumatoid arthritis of the hip appears to be in dispute since this disease is frequently considered both an indication and contra-indication.

The data obtained on results cannot be evaluated too satisfactorily since the questionnaire requested only the over-all results, without any references to the different reasons for which the operation was performed. It is now well known that the results after insertion of a prosthesis for fresh fracture are superior to results after the same procedure for rheumatoid arthritis of the hips. Thus, the results of this portion of the questionnaire are of limited value.

The functional results, in percentages, of the patients reported were: walking with one cane, 32.35 per cent; walking unaided, 28.8 per cent; walking with cane or crutches, or both, 20.8 per cent; not walking, and in a wheel chair, 10.1 per cent; and bedridden, 7.95 per cent. No information is available as to how many of the patients listed as walking with one cane are doing so on direct orders from their orthopaedic surgeon, even though they probably can walk unaided. Similarly, there is no information as to what percentage of the patients reported as not walking or bedridden after operation were not walking or were bedridden prior to the insertion of the prosthesis. These statistics are therefore of relatively little value.

An attempt was made in the questionnaire to evaluate the results in the opinion of the patients. However, in too many of the replies the doctors' and the patients' evaluation of the results were identical, suggesting that the patients' opinion was not actually asked and possibly that the patient was not really re-examined.

The evaluations of results according to both patient and doctor were: excellent—doctor, 24.2 per cent and patient, 26.3 per cent; good—doctor, 37.7 per cent and patient, 37.8 per cent; fair—doctor, 22.4 per cent and patient, 21.3 per cent; and poor—doctor, 15.7 per cent and patient, 14.6 per cent. In the opinion of The Committee giving this report these statistics are properly inaccurate.

The statistics concerning complications are recorded according to the number of men reporting and not as the actual number of complications:

In dislocation of the prosthesis, 111 surgeons reported anterior dislocation; 97, posterior; 72, lateral; and 41, central (through the acetabulum). In fractures as a result of insertion of the prosthesis, 178 surgeons reported early and 49 reported late fracture of the femur; and 8 surgeons reported fracture of the acetabulum.

Two hundred and fifty-four surgeons reported infection as a complication; 218, mortality; 157, phlebitis; 14, protrusion of the stem from the lateral aspect of the femur; 11, calcification in the capsule; 6, sciatic-nerve palsy; 5, pain; 2, ankylosis; and 2, nylon allergy.

The type and number of broken prostheses and the location of break were: Judet—41, stem and neck; 29, head; and 11, rim. Nylon Judet—9, head; and 2, stem. Naden-Reith—8, neck; and 5, stem. Eicher—14, stem; 10, neck angle; and

1, stem (bent). Collison—11, screws. Anderson—5, head; and 1, stem. Scuderi—5, stem. Lippman—4, stem. Moore—1, stem. Minneapolis—1, head. Jewett—1, central. McBride—1, neck. Girard—1, angle.

The relative frequency of breakage of the stem types of prosthesis is apparent. It is believed that all of the Eicher prostheses reported as breaking in this series were of the original stainless-steel type.

Data on the operative approach were recorded as to the number of times the approach was used. (It is the feeling of The Committee that in many instances the surgeon was not sure just what approach had been used.)

The posterior lateral approach was reported as used 3,736 times; the anterior, 1,709 times; and other approaches were used 503 times. The other approaches included: Watson-Jones, Moore, lateral, short Kocher, anterior lateral, transverse, posterior, and Osborn.

Additional data in respect to operative technique were rather unsatisfactory owing to the way the questions were worded. The following information was obtained:

1. The short rotators were frequently divided or sectioned.

2. Only about one in ten surgeons removed and subsequently replaced the trochanter.

3. The joint capsule was closed by 90 per cent of the surgeons, whereas the remaining 10 per cent closed the capsule in fresh fractures, but resected it in reconstructive procedures.

Total failures were reported in 756 instances. The surgical procedures performed to salvage some of the failures, and the results obtained were reported as follows:

The prosthesis was replaced with a smaller one in 41 with good results; prosthesis removed, 37 with fair to poor results; dislocation of prosthesis reduced, 26 with fair to good results; fusion of the hip, 24 with good results if fusion was obtained, but a high rate of pseudarthrosis; incision and drainage for infections, 20 with poor results; Colonna reconstruction, 14 with fair to good results; broken prostheses replaced, 5 with good results; obturator neurectomy, 4 with fair results; acetabulum deepened, 4 with fair results; and osteotomy, 2 with poor results. One Whitman reconstruction and one adductor tenotomy gave fair results and the result of one cup arthroplasty was poor.

Among the eighty-eight men who replied that they did not use hip prostheses, seventy-seven had not used this appliance because their practice was either limited to surgery of the hand, or because they were retired or physically disabled. The remaining eleven men had used a total of 119 prostheses and then discontinued their use because of poor results.

Committee for the Study of Femoral-Head Prostheses
Donald E. King, M.D.
Lee Ramsay Straub, M.D.
Claude N. Lambert, M.D., *Chairman*

DISCUSSION

Dr. Duncan C. McKeever, Houston, Texas: No doubt The Committee realizes many of the shortcomings of its current report. Some of the more obvious deficiencies could be responsible for the drop in the number of returns. It would seem to be a waste of time to collect the information requested when it cannot possibly be compiled into informative statistics.

The objective of The Committee is to determine whether a hip prosthesis is a feasible and useful procedure. The only cases of value in such a study are those in which a properly designed prosthesis is correctly inserted in a suitable hip.

No one has stated the principles to be followed in designing a prosthesis.

Some surgeons use one surgical approach, some another. No one has stated a list of necessary and desirable objectives by which the most suitable surgical technique could be determined.

No one has stated the anatomical, physiological, and psychological factors that have bearing on the success or failure of such a procedure.

No one has stated standards by which a proper course of postoperative care could be determined or when weight-bearing can be safely instituted.

Such standards, subject to later revision, could be formulated on the basis of present knowledge.

The following are some of the more obvious deficiencies in the questionnaire:

1. The question on the patient's age does not state whether the answer required is the age at the time of operation or the age at the time of check-up.

2. The questionnaire fails to take differences in diagnoses into account. So far as this report is concerned a hip prosthesis is a hip prosthesis regardless of the condition for which it is used. It is neither fair nor informative to compare the result obtained after a prosthesis is applied in a fresh fracture with the result obtained after a prosthesis is applied in an old fracture, a malum coxae senilis, or in any other condition. These conditions should be evaluated separately.

3. There is a listing for excellent, good, fair, and poor results. There is no listing for an out-and-out failure. There is no differentiation for a prosthesis inserted in a bedridden patient, a procedure done merely to relieve pain and enable the patient to sit and move about in bed. If this goal is attained the result is excellent. However, such a result would be classified a failure if the patient had been expected to walk.

4. There is no provision for comparison of quality of results in relation to duration of results. A hip prosthesis may give an excellent result at the end of one year, a good result at the end of five years, a poor result at the end of eight years, and then be a failure at the end of ten years. A patient who walked for one year after insertion of a hip prosthesis, without symptoms or without even a limp and then died of some other condition would be considered as having an excellent result, whereas, in another few months or years osteoporosis or intrapelvic protrusion might have occurred and the result would have been classified as a complete failure.

5. There is no differentiation in the fair or poor results, or failures, as to whether or not they occurred as a result of a fault or failure of the prosthesis, a fault or failure of the surgeon, or a fault of the patient's metabolic state. There could, in many cases, be a combination of these reasons. Such failures cannot be used to condemn prostheses *per se*.

6. In the number of months of follow-up there is a minimum, a maximum, and an average. In my series of cases a one-month follow-up is listed along with a 114-month follow-up. This gives an average of 57.5 months. Of what significance is this figure? Does multiplication of such figures make them more significant?

7. Is any bilateral case to be compared with any unilateral case? Is a procedure done for bilateral malum coxae senilis to be compared with a bilateral procedure done for Strümpell-Marie arthritis, or bilateral congenital dislocation of the hip in an achondroplastic dwarf? On what basis?

It would appear that a complete revision of the basic questionnaire, and the formulation of some fundamental standards for assessment of cases must be carried out before useful and informative statistics can be compiled.

A Clinical Comparative Study of Autogenous and Homogenous-Bone Grafts

BY PETER L. CARNESALE, M.D., AND JACK D. SPANKUS, M.D., MILWAUKEE, WISCONSIN

From the Orthopaedic Surgical Service, Wood Veterans Administration Hospital, and the Marquette University School of Medicine, Milwaukee

Many opinions have been expressed concerning the clinical success of homogenous or autogenous-bone grafts [1-27]. The use of the autogenous graft is popular because most surgeons feel that it is superior. We will substantiate this impression through the clinical analysis of 304 bone-grafting procedures. No similar comprehensive comparative study of autogenous and homogenous-bone grafts has been reported. These procedures were done at the same hospital under the direction of the same supervising orthopaedic staff—the optimum condition for the most reliable comparative conclusions. Reports have been made by investigators at different institutions on the results of either autogenous or homogenous-bone-grafting, but comparing the results of one group with those of another is unreliable because of the many variables.

Only two comparative clinical studies are reported in the literature. In 1949, Weaver reported seventeen cases in which fresh homogenous bone was used and thirty-two cases in which autogenous bone was used. He had an 8.8 per cent incidence of failure with fresh homogenous grafts as compared with a 21.9 per cent incidence of failure with autogenous grafts. He suggested that his success with fresh homogenous bone was due to the availability of a larger quantity of the particular type of bone desired. In 1954, Hay noted "little difference" between autogenous and homogenous grafts in a series of fifty-five bone-graft procedures, in thirty-three of which autogenous and in twenty-two of which homogenous bone was used.

We are reporting a study of 143 consecutive bone-grafting procedures in which merthiolate-preserved, homogenous bone was used, 137 consecutive bone-grafting procedures in which autogenous bone alone was used, and twenty-four consecutive bone-grafting procedures in which a mixture of merthiolate bank bone and autogenous bone was used. These figures do not include ten procedures in which there was no follow-up. All bone-grafting procedures done between January 1948 and January 1958 are included. The age of the patients ranged from nineteen to sixty-two years. The use of merthiolate-preserved, homogenous bone was begun in March 1949, and the method of preservation was that described by Reynolds. This method was found to be simple and inexpensive; no elaborate equipment was necessary.

The incidence of infection for autogenous grafts was 9 per cent (thirteen cases) but in one case there was drainage at the time of bone-grafting. The incidence of infection for homogenous grafts was 5 per cent (seven cases) but in two cases there was drainage at the time of surgery. For the combined grafts the incidence of infection was 8 per cent (two cases). The total incidence of postoperative infection was 7 per cent (twenty-two cases).

We used merthiolate bone indiscriminately in all cases early in the series. Soon we became more selective because we realized more failures and eventually used homogenous bone only in specific situations. For example, it was used when the available supply of bone was not adequate, when the hazard of surgery was thought to be too great to obtain autogenous bone, when no previous plan was made to obtain autogenous bone, and, often, when a mechanical support was necessary as in a depressed plateau fracture of the tibia.

The following conclusions were drawn by several investigators after evaluating autogenous and homogenous grafts in experimental animals [3,4,10,11,15,18,19,21,24]:

No one has stated standards by which a proper course of postoperative care could be determined or when weight-bearing can be safely instituted.

Such standards, subject to later revision, could be formulated on the basis of present knowledge.

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The following conclusions were drawn by several investigators after evaluating autogenous and homogenous grafts in experimental animals [3,4,10,11,15,18,19,21,24]:

TABLE I

AN ANALYSIS OF THE RESULTS AS TO THE TYPE OF GRAFT EMPLOYED

Type of Bone	Total	Failure (*No.*)	Failure (*Per cent*)
Autogenous-bone graft			
Block graft	54	9	17
Chip graft	83	7	8
Homogenous-bone graft			
Block graft	48	15	31
Chip graft	95	18	19
Autogenous and homogenous graft			
Autogenous-block and homogenous-chip graft	6	2	33
Autogenous-chip and homogenous-block graft	1		
Autogenous and homogenous-chip graft	17	3	18
Total	304*	54	

* Ten cases without follow-up not included.

1. There is a negligible difference between frozen, freeze-dried, and merthiolate-preserved homogenous bone.

2. Homogenous bone is slower to incorporate, causes more tissue reaction, and has a higher incidence of failure than autogenous bone.

A procedure was called successful when there was clinical and roentgenographic evidence of incorporation of the graft. The grafts listed in Table I were used either in the form of a full-thickness block or in the form of thin wafers, chips, or bone meal. The latter three are grouped together. Chip grafts were made from cancellous bone obtained with a rongeur, curette, or bone cutter. Bone meal was made by passing cancellous bone through a bone mill. No histological investigation was made concerning the host-tissue reaction to homogenous grafts.

Table II summarized all the conditions for which bone grafts were used. Tables III, IV, V, and VI give in more detail the information listed in Table II. The subsequent comparative review of autogenous and homogenous grafts will refer to these tables. The number of cases of various types of procedure is small in many instances. It is not the intent of this paper to draw conclusions from them alone. The significant conclusions are obtained by comparing the total figures of each type of bone graft.

Spine Fusions (Seventy-Three Cases; Table II)

It may be noted that all conditions, except tuberculosis, which necessitated arthrodesis of one or more segments of the spine are grouped together. As stated previously, a fusion was called successful when there was clinical and roentgenographic evidence of incorporation of the graft. Solid union of the transplant across one interspace and a pseudarthrosis at another was considered to be a failure. The highest incidence of pseudarthrosis occurred when homogenous bone alone was used. *Merthiolate bank bone* in Table II is listed separately because the graft bone was predominantly bank bone although fragments of autogenous spinous processes and laminae were included. The heading in Table II of *Autogenous and Bank Bone* refers to fusions in which bank bone supplemented bone taken from the ilium. Here the bank bone was not predominant. Of the thirty-four spine fusions with homogenous bone, twelve were performed with a full-thickness H homograft and twenty-two with homogenous chips. There were four failures in each group.

A point of interest is that spine fusion for tuberculosis was more successful than for any other condition. All fusions were successful in this group. This was attributed to the long period of immobilization and bed rest.

TABLE II

AN ANALYSIS OF THE RESULTS OF BONE GRAFTS IN SPECIFIC CONDITIONS UTILIZING AUTOGENOUS BONE AND MERTHIOLATE BANK BONE

Indication	Autogenous Bone			Merthiolate Bank Bone			Autogenous and Bank Bone			Total No. Procedures*
	Total	Failures	Failed (*Per cent*)	Total	Failures	Failed (*Per cent*)	Total	Failures	Failed (*Per cent*)	
I. Arthrodesis										
A. Spine										
1. Low-back pain (unstable back, arthritis, spondylolisthesis, herniated nucleus pulposus)	11	1	9	34	8	24	10	1	10	55
2. Tuberculosis	7			9			1			17
3. Cervical dislocation				1						1
B. Other joints										
1. Tuberculosis	11	1	9	2	2	100	1	1	100	14
2. Other arthropathies	49	3	6	15	3	20	3			67
3. Flail joint due to paralysis from poliomyelitis or injury	7			5	3	60	1			13
II. Fresh fractures	3			24	2	8				27
III. Bone grafts for correction										
A. Ununited fractures	39	10	26	31	9	29	8	3	38	78
B. Malunion	4			3						7
C. Defects caused by:										
1. Osteomyelitis	5	1	20	5	2	40				10
2. Benign tumors and cysts	1			10	3	30				11
3. Malignant tumors				4	1	25				4
Total	137	16	12	143	33	23	24	5	21	304*

*Ten cases without follow-up not included.

TABLE III

AN ANALYSIS OF THE RESULTS OF THE USE OF BONE GRAFTS IN ARTHRODESIS OF JOINTS OTHER THAN THE SPINE (94 CASES)

Indication	Autogenous Bone		Merthiolate Bank Bone		Autogenous and Bank Bone	
	Successful	Failures	Successful	Failures	Successful	Failures
Tuberculosis						
Wrist	4			1		
Hip	3			1		1
Knee	1					
Ankle	2					
Subastragalar		1				
Total	10	1		2		1
Other arthropathies						
Shoulder joint						
Traumatic arthritis			2	1		
Elbow joint						
Traumatic arthritis	1		1		1	
Wrist joint						
Ununited navicular fracture	7		2	2	1	
Traumatic arthritis	1					
Subsequent to removal of lunate	1	1				
Ununited lunate fracture	1					
Avascular necrosis of lunate	1					
Quiescent rheumatoid arthritis	1					
Hip joint						
Malum coxae senilis	6	1	1		1	
Traumatic arthritis	3					
Avascular necrosis, femoral head	2		1			
Pyarthrosis (quiescent)	1					
Sacro-iliac joint						
Traumatic arthritis	3					
Ankle joint						
Traumatic arthritis	15	1				
Subastragalar joint						
Traumatic arthritis	2		4			
Tarsometatarsal joint						
Traumatic arthritis	1		1			
Total	46	3	12	3	3	
Flail joint from polio or injury						
Shoulder	1		1	3	1	
Wrist	2					
Ankle	2					
Ankle and subastragalar	1		1			
Tibiocalcaneus	1					
Total	7		2	3	1	
Total	63	4	14	8	4	1

Arthrodesis of Tuberculous Joints (Except Vertebral) (Fourteen Cases; Table III)

Arthrodesis of tuberculous joints, other than those of the spine, failed when homogenous bone was used (three cases). An onlay graft across the wrist in one case failed to incorporate at its distal end. However, the patient remained asymptomatic, and further surgery was not necessary. One Brittain fusion of the hip in which a full-thickness homograft was used failed. Another Brittain hip fusion in which a combination of autogenous and homogenous bone was used failed.

TABLE IV

AN ANALYSIS OF THE RESULTS OF THE USE OF BONE GRAFTS IN FRESH FRACTURES (27 Cases)

Site of Fracture	Autogenous Bone		Merthiolate Bank Bone	
	Successful	Failures	Successful	Failures
Ulna	1			
Femoral neck				1
Trochanteric area	2		5	1
Femoral shaft			1	
Tibial plateau			7	
Tibial shaft			1	
Calcaneus			8	
Total	3	0	22	2

TABLE V

AN ANALYSIS OF THE USE OF BONE GRAFTS IN UNUNITED FRACTURES (78 Cases)

Site of Fracture	Autogenous Bone		Merthiolate Bank Bone		Autogenous and Bank Bone	
	Successful	Failures	Successful	Failures	Successful	Failures
Humerus	1			2	2	
Radius	1	1				
Ulna	5	1	1			
Carpal navicular	1					
Femoral neck			1	1		
Trochanteric area	1		2			
Femoral shaft	3	1	7	3	2	1
Tibia	15	7	11	3	1	2
Medial malleolus	2					
Total	29	10	22	9	5	3

Arthrodesis for Arthropathies Other Than Tuberculosis (Sixty-Seven Cases; Table III)

In the series of homogenous-bone-graft procedures two failures occurred in the same patient after two attempts had been made to fuse a wrist with homogenous-bone chips for a painful joint resulting from an ununited navicular fracture. Success was obtained after a sliding graft was used. The third failure occurred after an extra-articular shoulder arthrodesis of the Brittain type with a homogenous strut.

In the series of autogenous-bone-graft procedures a pseudarthrosis developed at the distal end of a sliding graft of the wrist which was fused because of pain due to avascular necrosis of the lunate bone. There was a failure to fuse an ankle joint after the use of an onlay autogenous graft. A pseudarthrosis resulted from the use of an autogenous full-thickness graft in a Badgley hip fusion.

Arthrodesis of Flail Joints Due to Poliomyelitis or Injury (Thirteen Cases; Table III)

Three failures in the series of homogenous-bone-graft procedures involved the shoulder joint. In one patient, two attempts were made to do a Brittain type of fusion with a homogenous strut. The graft failed to unite with the humerus. After an intra-articular fusion was done the graft incorporated. The third failure occurred in a similar procedure when the strut became displaced ten days after operation.

TABLE VI

AN ANALYSIS OF THE RESULTS OF FILLING BONE DEFECTS WITH BONE (25 Cases)

Etiology of Defect	No.	Autogenous Bone		Merthiolate Bank Bone	
		Successful	Failures	Successful	Failures
Osteomyelitis	10	4	1	3	2
Tumors and cysts	15				
Benign (11 cases)					
Bone cyst		1		1	1
Giant-cell tumor				3	1
Enchondroma				2	
Fibroma					1
Neuroma				1	
Malignant (4 cases)					
Multiple myeloma				3	
Sclerosing osteogenic sarcoma					1
Total	25	5	1	13	6

Fresh Fractures (Twenty-Seven Cases; Table IV)

The majority of the grafts were used to correct depressed fractures of the tibial plateau and the calcaneus. These homogenous grafts consisted of small blocks of bone inserted to maintain elevation of the depressed fragments. Subtrochanteric osteotomies which were produced for treatment of various conditions were listed as fractures in the trochanteric area. One non-union occurred at an osteotomy site when homogenous bone was used. An attempt to treat a femoral-neck fracture by inserting a homogenous block of bone three and a half inches by a quarter inch through the neck failed when the graft did not incorporate.

Ununited Fractures (Seventy-Eight Cases; Table V)

Seven of the ten failures in the group of autogenous-bone-graft procedures were caused by postoperative infection. The fractures had been compound wounds or had prior surgical intervention with resultant postoperative drainage. Two other failures occurred in ununited fractures of the ulna and the radius. The tenth failure resulted from the use of autogenous chips in an ununited supracondylar fracture of the femur.

Two of the nine failures in the series of homogenous-bone-graft procedures were caused by postoperative infections. The use of homogenous chips for two ununited supracondylar fractures of the femur resulted in failure. The fifth failure occurred when the distal end of an onlay homogenous graft did not incorporate in an ununited fracture of the shaft of the femur. Another failure resulted when an onlay homogenous graft broke because of early mobilization of an ununited fracture of the humerus. In a similar case another onlay graft used for a non-union failed to incorporate at the distal end of the humerus. Homogenous chips which became incorporated in the neck of the femur, but did not stop the progression of avascular necrosis of the femoral head, were responsible for the eighth failure. In the ninth case a pseudarthrosis developed after homogenous chips had been inserted in an ununited fracture of the tibia.

Three failures resulted after the use of a combination of autogenous and homogenous bone: two because of postoperative infection, the third because of non-union of a supracondylar fracture of the femur.

Four of the failures previously described resulting from non-union of a supracondylar fracture of the femur occurred in the same patient.

Malunions (Seven Cases; Table II)

All cases of bone-grafting resulted successfully.

Defects Caused by Osteomyelitis (Ten Cases; Table VI)

Two of five attempts to fill defects in bone due to active chronic osteomyelitis after saucerization and the insertion of homogenous bone failed. One of the successful results in the homogenous bone-graft series was in a patient with a sclerosing osteitis. One of five cases resulted in failure when autogenous chips were used.

Defects Caused by Tumors (Fifteen Cases; Table VI)

The following are our failures: A bone cyst of the femur packed with homogenous-bone chips resulted in failure because the patient fell and fractured the femur through the cyst twenty-three days after surgery. A giant-cell tumor of the calcaneus was packed with homogenous chips after curettement of the defect; one year later the tumor recurred. At subsequent surgery there was no evidence of incorporation of the graft. A fibroma of the distal femur was removed by curettement, and the defect was packed with homogenous-bone chips; five months later there was roentgenographic evidence of absorption. Study of another biopsy specimen resulted in the diagnosis of reticulum-cell sarcoma. An attempt was made to resect a sclerosing osteogenic sarcoma of the upper end of the tibia and to fuse the tibia to the femur with the use of anterior and posterior onlay homogenous grafts. The graft failed to incorporate and the tumor recurred.

One patient with multiple myeloma of eleven years' duration was previously reported [9]. He had a defect in the shaft of the femur and one in both tibiae. Massive onlay homogenous grafts were used for the defects of the femur and of one tibia. Homogenous chips were used for the defect of the other tibia. These grafts incorporated and the patient is still active and working.

SUMMARY

In the past ten years, 137 autogenous and 143 merthiolate-preserved, homogenous-bone-grafting procedures have been performed. Twenty-four additional procedures were done in which a combination of autogenous and homogenous-bone grafts was used. Thus there is a total of 304 surgical procedures. There were sixteen (12 per cent) failures in the autogenous group, thirty-three (23 per cent) in the homogenous group, and five (21 per cent) in the group in which a combination of autogenous and homogenous-bone grafts were used. All failures were included regardless of the reason, and no distinction was made as to the etiology of the nonunion.

The incidence of failure with autogenous chips was 8 per cent. When a full-thickness autogenous-bone block was used, the incidence of failure was 17 per cent. The incidence of failure with homogenous chips was 19 per cent; when a full-thickness block graft was used the incidence was 31 per cent. A combination of autogenous and homogenous chips resulted in an 18 per cent incidence of failure. The occurrence of infection with merthiolate-preserved homogenous grafts was less than that with autogenous grafts. Bone grafts inserted in the presence of infection usually resulted in failure. No allergic reaction or sensitivity to merthiolate occurred unless one concludes that the greater number of failures from the use of merthiolate bone is on this basis.

CONCLUSION

This clinical review confirms the impression that autogenous grafts are superior to homogenous grafts. Homogenous-bone grafts are satisfactory in specific situations, but they must be protected for a longer period of time than autogenous grafts. They may be used when the available supply of autogenous bone is not adequate for the particular situation or when the hazard of surgery is materially increased by the obtaining of autogenous bone. Homogenous bone may be used also when no previous plans have been made to obtain autogenous bone. Homogenous bone was often used when a mechanical support was necessary and the condition did not

warrant the taking of an autogenous graft, for example, in a depressed plateau fracture of the tibia. We believe that it is of the most value in supplementing autogenous bone.

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Treatment of Giant-Cell Tumor of Bone*

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In 1956, Lichtenstein said that the more he saw of giant-cell tumors of bone, the more wholesome was his respect for them. Jaffe, in 1953, said that giant-cell tumor of bone was a treacherous lesion that was difficult to assay in respect to its clinical behavior. These two comments, by recognized authorities in the field of bone tumors, give a thumbnail sketch of the problem presented by giant-cell tumor. For some time we have thought that the treatment of giant-cell tumors leaves much to be desired, and the analyses of several large series of cases that have appeared in the literature serve only to add to the confusion of the reader when he attempts to determine the proper course of treatment for any given giant-cell tumor of bone. It was because of these conflicting claims and because of the confusion concerning the best method of treatment mentioned in the literature as well as our own ignorance of the best therapy that this study was undertaken. We thought that thorough study of a large series of giant-cell tumors of bone in which long-term follow-up data were available would be of value.

For reviews of the literature on the entire problem of giant-cell tumor the reader is referred to the articles by Coley, Higinbotham and Kogure, by Stewart and Richardson, by Williams, Dahlin and Ghormley, and by Jaffe [6].

MATERIAL

The series consisted of 116 consecutive cases of giant-cell tumor, taken from the files of the Mayo Clinic. Variants of giant-cell tumor were carefully excluded because of their notorious dissimilarity to giant-cell tumor in regard to response to therapy. By inquiries directed to patients, referring physicians, and relatives, follow-up information for varying lengths of time on every patient was obtained. This study is concerned primarily with determination of the results of the prevailing methods of treatment of these tumors.

The problem of definition of recurrence developed when the case records were reviewed. The roentgenograms and other available data sometimes failed to disclose whether further treatment was given because of actual or suspected recurrence. Prior irradiation or surgical treatment often made the interpretation of the roentgenographic findings very difficult. Sometimes, additional radiation therapy was given for recurrent pain or disability when no appreciable change could be seen on the roentgenograms. For the purposes of this study, a recurrence was considered to have taken place when the patient received any treatment three months or more after completion of the initial therapy. A successful case was one in which no recurrence was noted after initial therapy.

The ages of the definitive group of 116 patients varied from twelve to seventy-one years, and 63.8 per cent were in the third and fourth decades of life. Women predominated in the ratio of sixty-four to fifty-two (Table I).

Sixty-three of the tumors occurred in the upper portion of the tibia or the lower portion of the femur, with the latter site being the most common. The third most common site was the sacrum, with eleven tumors, and the fourth, the lower portion of the radius, with nine (Fig. 1).

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Since many of the patients received multiple forms of treatment, they were all grouped arbitrarily into five categories according to the primary treatment given as follows: (1) patients treated by curettage, (2) patients treated by excision, in which the entire tumor and its encasing shell of bone were removed, (3) patients treated by amputation, (4) patients treated by irradiation, and (5) two patients who fitted into none of the preceding categories. One of these had biopsy and aspiration only, and the other had biopsy and Coley's toxin; both died.

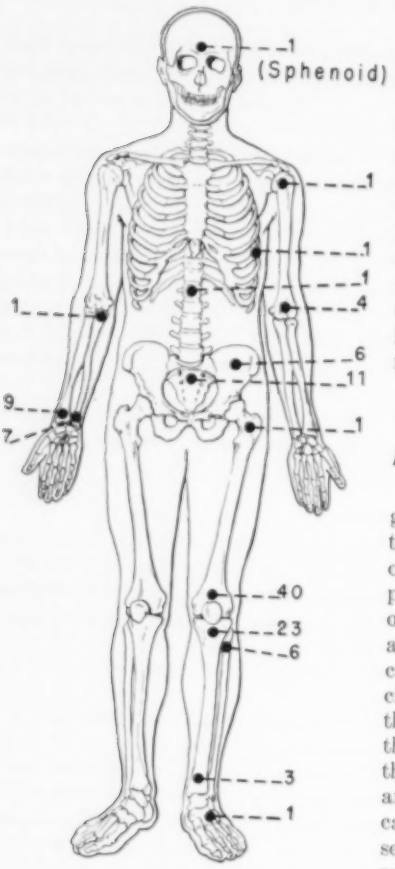

FIG. 1
Skeletal distribution of 116 cases of giant-cell tumor.

Since many of these patients received various forms of therapy either primarily or for recurrences of the tumor, such as excision or amputation after curettage or irradiation, the same patients are of necessity considered in more than one treatment category. This has made the clear and concise presentation of the results of therapy for each of the 116 patients virtually impossible. However, it is the purpose of this study to present the results of the various forms of therapy rather than to present case histories that include all phases of treatment for each patient.

OBSERVATIONS ON TYPES OF TREATMENT

Patients Treated by Curettage

The seventy-one patients in this group were subjected to 146 separate treatment sessions. Many combinations of treatment were employed for these patients including curettage with or without grafts, preoperative and postoperative radiation therapy, and chemical cautery of the cavity. None of these ancillary measures appeared to influence the rate of recurrence in this group, although the numbers of patients receiving the various combinations of treatment are too small to be statistically significant in many instances. The data on age, sex, and skeletal localization for these seventy-one patients did not deviate significantly from those for the entire series. The patients treated primarily by curettage were further subgrouped according to the length of time they were observed (Table II).

A solitary pulmonary metastatic deposit developed in one of these patients four and one-half years after the primary lesion in the distal portion of the radius had been controlled by curettage and roentgen therapy. The primary tumor and the excised metastatic deposit were histologically benign and not different from the other 115 originally benign giant-cell tumors in this series that did not metastasize.

Amputation became necessary as a secondary procedure in fourteen of the patients treated by primary curettage. Secondary sarcoma, massive recurrence, and persistent draining and disabling lesions were the reasons for amputation.

Successful results from curettage—that is, freedom from recurrence during the period of follow-up—were obtained in only thirty of the seventy-one patients.

TABLE I

AGE AND DISTRIBUTION OF 116 PATIENTS WITH GIANT-CELL TUMOR OF BONE *

Age (*Years*)	Sex (*No. of Patients*)		Total No. of Patients
	Male	Female	
0- 9			
10-19	3	7	10
20-29	18	23	41
30-39	16	17	33
40-49	10	13	23
50-59	3	3	6
60-69	2		2
70-79		1	1
Total	52	64	116

* The youngest patient was twelve years of age; the oldest was seventy-one.

TABLE II

RESULTS IN PATIENTS TREATED BY CURETTAGE

Follow-up Period (*Years*)	(*Mean Years*)	Site of Tumor	Results		No. of Patients
			Success	Failure	
0-4	2.4	Upper tibia	3	1	
		Sphenoid	1		
		Lower femur	2	4	
		Spine (body L-1)		1	
		Lower radius		1	
		Sacrum		1	
		Upper fibula		1	
					15
5-9	7.0	Upper tibia	2	2	
		Lower femur	1	3	
		Sacrum	1	2	
		Upper fibula	1		
		Ilium		1	
		Lower humerus		1	
		Lower radius		1	
					15
10-14	12.3	Lower femur	3	3	
		Lower tibia	1		
		Lower humerus	1	1	
		Upper tibia		2	
		Ilium		2	
		Lower radius		1	
					14
15-19	17.1	Upper tibia	2	1	
		Lower femur	1	1	
		Upper femur	1		
		Ilium	1		
		Sacrum	1	1	
		Lower tibia		1	
		Lower radius		1	
					11
>20	30.6	Lower femur	4	3	
		Foot	1		
		Sacrum	1		
		Upper tibia	1	3	
		Lower ulna	1		
		Lower humerus		1	
		Lower radius		1	
					16
				Total	71

In the remaining forty-one patients the tumor was judged to have recurred because additional treatment was considered necessary three or more months after completion of the primary therapy. Some of these patients had as many as four recurrences, but the majority had only one or two. It may be seen in Figure 2 that 70.4 per cent of the seventy-one patients were free of recurrence at the end of one year, but when they were followed for five years this figure dropped to 45.9 per cent, and at ten years it was only 40.4 per cent. There were no recurrences after ten years. This first computation of the recurrence rate includes patients who had had some

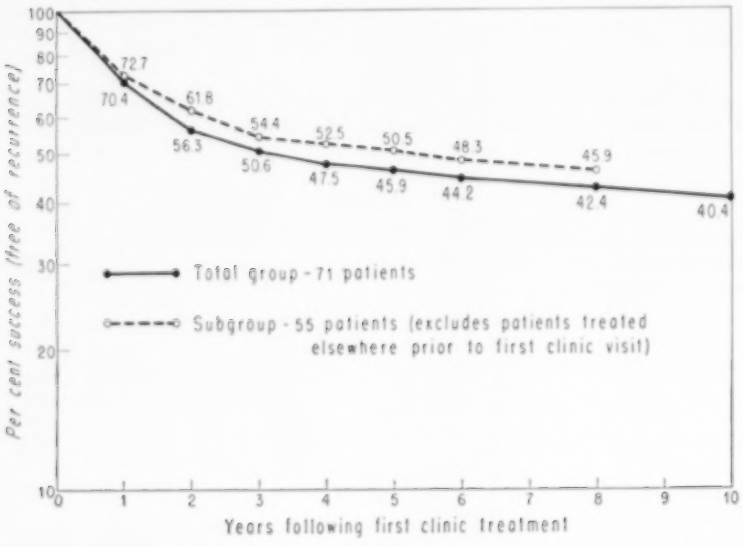

FIG. 2

Percentage of patients treated by curettage who were free of recurrence of giant-cell tumor at stated intervals after the first treatment at the Clinic.

of their treatment elsewhere, but the recurrence data as shown on the graph are formulated from the date of the first treatment at the Mayo Clinic. The second computation of the recurrence rate on the fifty-five patients who had their first treatment at the Mayo Clinic (that is, patients exclusive of the sixteen who had had their initial treatment elsewhere) revealed that 50.5 per cent were free of recurrence after five years, and none had recurrence after eight years. This smaller group is represented by the second curve in Figure 2.

The distribution of patients according to interval between the first treatment and recurrence or recurrences is shown in Tables III through V. Twenty-one of the forty-one patients who had a first recurrence had it within the first year, and another ten patients had it in the second year after completion of the primary treatment. Six of the sixteen patients who had a second recurrence had it within a year after treatment of the first recurrence, whereas eleven patients had the second recurrence within two years after treatment for the first recurrence. All but one of the seven patients who suffered a third recurrence manifested it within two years after treatment for the second recurrence. It is apparent that most recurrences, whether first, second, or third, developed at some time within the first two years after previous therapy, although some were long delayed; about half became manifest within one year.

Patients Treated by Excision

The tumors of fourteen patients were treated primarily by excision, and those

TABLE III

DISTRIBUTION OF PATIENTS HAVING ONE OR MORE RECURRENCES AFTER FIRST TREATMENT AT MAYO CLINIC

Interval between First Treatment and First Recurrence	No. of Patients
0-2 months	
3-5 months	4
6-8 months	9
9-11 months	8
1-2 years	10
2-3 years	4
3-4 years	2
4-5 years	1
5-6 years	1
6-7 years	
7-8 years	1
8-9 years	
9-10 years	1
Total	41

of another four were completely excised after failure of curettage and ancillary measures. The ages of the fourteen patients varied from eighteen to forty-eight years; eight patients were women. The tumors were situated as follows: in the lower portion of the ulna in five patients; in the lower portion of the radius in three; in the upper portion of the fibula in three; in the lower portion of the femur in one; in the upper portion of the femur in one; and in the upper portion of the tibia in one.

Twelve of the fourteen patients had no recurrence while being followed for an average of 16.2 years; six of these, however, died from unrelated causes. Two had unsuccessful results. In one of these, excision of a tumor in the upper portion of the femur was probably successful, but ancillary radiation therapy was given, and the functional result was compromised by radiation necrosis of the acetabulum. The other patient had non-union after excision of a tumor of the upper end of the tibia, and secondary amputation was necessary. None of the four tumors excised after unsuccessful prior therapy has recurred. Two of these patients were followed for six and forty-eight years, respectively, after excision, whereas the other two were observed for less than one year.

The significant fact is that sixteen of the eighteen patients treated by excision of the tumor and its surrounding shell of bone and fibrous tissue have had successful results, and the follow-up periods have averaged 14.9 years. In one of these successfully excised tumors, anaplastic fibrosarcoma coexisted with the giant-cell tumor in the lower portion of the femur but the patient was alive and well forty-seven years later.

Patients Treated by Radiation

Irradiation after biopsy was the primary form of treatment in thirteen patients whose ages varied from twenty-one to fifty-five years. Six patients were women. Three of the tumors occurred in the sacrum, four in the lower portion of the femur, and one each in the ilium, the upper portion of the humerus, the lower portion of the tibia, the upper portion of the tibia, the lower portion of the radius, and the seventh rib. The tumor of the ilium was associated with generalized Paget's disease of bone which involved the same ilium as did the giant-cell tumor.

Irradiation was not successful in controlling the tumor in any of these thirteen patients. Eight patients were followed for periods that varied from five to thirty years (average, seventeen years) after this form of primary therapy, and there were thirty-six individual trials of treatment. One patient died nineteen years

after initial treatment when a postoperative meningitis developed after operation for recurrence. Two patients required amputation because of recurrent tumor, and one required it because of a pathological fracture which appeared to be the result of both recurrence of the tumor and radiation necrosis. The remaining patients were treated with curettage and bone-grafting.

Five of the thirteen patients were followed for less than five years; four died within this period, and one was lost to follow-up. No post-mortem examinations were obtained on the four patients who died, but their local physicians reported that in two with sacral tumors and in one with a rib tumor the cause of death appeared to be due to recurrence, whereas, in the remaining patient, death was caused by "pulmonary trouble" of an unknown type.

TABLE IV

DISTRIBUTION OF PATIENTS HAVING TWO OR MORE RECURRENCES AFTER FIRST TREATMENT AT MAYO CLINIC

Interval between First and Second Recurrence	No. of Patients
0-2 months	1
3-5 months	3
6-8 months	2
9-11 months	
1-2 years	5
2-3 years	
3-4 years	
4-5 years	2
5-6 years	
6-7 years	2
7-8 years	
8-9 years	1
Total	16

TABLE V

DISTRIBUTION OF PATIENTS HAVING THREE OR MORE RECURRENCES AFTER FIRST TREATMENT AT MAYO CLINIC

Interval between Second and Third Recurrence	No. of Patients
0-2 months	
3-5 months	2
5-8 months	
9-11 months	2
1-2 years	2
2-3 years	
3-4 years	
4-5 years	
5-6 years	1
Total	7

Radiation therapy was employed at some time for seventy-four of the 116 giant-cell tumors. Its effect was usually difficult to evaluate, as Stewart and Richardson have also noted. In some instances, sloughing of the wound, radiation dermatitis, atrophy of the extremity, and persistent drainage from the tumor site occurred after its use. In at least one case these side effects necessitated amputation. In another, chronic postradiation ulceration over the sacrum contributed much to the patient's morbidity and disability. Various methods of radiation were employed, including implantation of radon seed, application of radium, and roentgen therapy. Little justification for either preoperative or postoperative irradiation is

evident here, except for tumors so situated that adequate surgical treatment is impossible.

Patients Treated by Amputation

Amputation was the primary form of therapy in sixteen cases. Reasons for amputation included mistaken diagnosis of malignant disease (in the early years covered by this study), extensive destruction of bone, severe disability, infection, and hemorrhage. In one patient, who lived for twenty-three years after amputation, a fibrosarcoma coexisted with the giant-cell tumor within the same radiolucent area. One patient of this group was lost to follow-up, and one died from pneumonia eight years after amputation, but the other patients were alive and well when last heard from. Four received irradiation of the stump and groin after operation, and one received irradiation of the tumor before operation.

Fourteen of the sixteen patients were followed for more than five years (average 20.6 years). Three of the fourteen patients died—two from heart disease, and one from pneumonia. In none was a recurrence noted. Two patients were followed for less than five years; one of these was lost to follow-up, and the other had no recurrence at two and one-half years after the first type of therapy.

Eighteen patients underwent amputation after primary treatment failed. The primary treatment was curettage in fourteen, excision in one, and radiation in three. Ten were followed for five years or more (average, 26.7 years), seven were followed for less than five years, and one was lost to follow-up. Eight patients underwent amputation because of malignant transformation, and ten because of massive recurrence, progression of the tumor, or drainage. There was no recurrence in these last ten patients during the follow-up period. The patients with malignant disease are considered separately.

OBSERVATIONS ON RESULTS AFTER TREATMENT

Sarcomatous Transformation

A total of ten sarcomata developed after treatment for giant-cell tumors which were originally no more anaplastic than the remainder of the 116 lesions. Eight of these malignant tumors were fibrosarcomata and two were osteogenic sarcomata. Nine tumors had received radiation along with other therapy for the original benign lesion. One sarcoma developed seventeen months after curettage and grafting for a giant-cell tumor of the lower end of the femur, and produced death within a year despite amputation.

Eight of the nine giant-cell tumors undergoing sarcomatous change after radiation therapy in this series were reported previously by Sabanas and Dahlin. One of the nine cases reported by these authors was excluded from the present series because of a change in diagnosis. The ninth case included here is that of a lesion in the sacrum which has been followed for only nine months.

The intervals between primary treatment and the proof of sarcomatous change in these nine patients varied from forty-three to 167 months. In all but three, the interval was five or more years. One of these sarcomata occurred in the ilium and caused death in less than a year. Another arose in the sacrum of a patient who was still alive nine months after the diagnosis was established. Major tubular bones were affected in eight instances: five sarcomata were in the lower end of the femur, two were in the upper end of the tibia, and one was in the lower end of the humerus. All eight of the affected extremities were amputated, with the result that six of the patients died within three years from the effects of the malignant tumor and two survived for intervals of twenty-four and seventeen years, respectively, without evidence of recurrence.

In addition to these ten sarcomata which developed after treatment of giant-cell tumors, there were two instances in which sarcoma and giant-cell neoplasm coexisted in the same radiolucent area at the time of the first treatment. In both

FIG. 3-A FIG. 3-B FIG. 3-C

Figs. 3-A through 3-C: Giant-cell tumor of the distal part of the right radius in a woman, twenty-eight years old, who had an unsuccessful trial of radiation therapy in 1951, with recurrence of the tumor.

Fig. 3-A: Appearance in July, 1952.

Fig. 3-B (top): The tumor presenting through the dorsal cortex of the radius at operation.

Fig. 3-B (bottom): Portion of the right radius removed; the defect was bridged with dual tibial grafts.

Fig. 3-C: Solid arthrodesis evident in January 1955. (At the time of writing the patient worked as a typist, and had minimal disability. The cosmetic appearance is good.)

of these, as previously mentioned, the condition had been cured for long intervals—one by total excision of the lesion, and one by amputation.

Giant-Cell Tumor of the Sacrum, Ilium, and Vertebra

We were especially careful in our attempts to avoid inclusion of variants of giant-cell tumor in these bones because of the known rarity of giant-cell tumor here. Nevertheless, this series contains eleven sacral, six iliac, and a single vertebral giant-cell tumor, the last affecting the first lumbar vertebra. Except for one iliac lesion which was treated with Coley's toxin alone and one sacral lesion, all these lesions received radiation therapy. Because of their locations, only partial removal of many of them was possible. Despite this, five patients with sacral and three with iliac giant-cell tumor were alive and well for intervals that varied from five to thirty-five years. The remaining patients were either dying or had died from the effects of their tumors, although some did live usefully for a number of years. Two of these patients were among those who had postirradiation sarcomata. It seems likely that irradiation was responsible for some of the "cures" and for some of the prolongation of life in patients with sacral, iliac, and vertebral giant-cell tumors.

Grading of Giant-Cell Tumors

The original tumors in this series were carefully studied to find out if the histological appearance would enable estimation of whether the tumor would recur or undergo subsequent malignant transformation. The tumors that were cured by one treatment, that recurred, that ultimately became malignant, and even the one that metastasized although histologically benign, were indistinguishable. As Troup has shown, those giant-cell tumors that contain frankly sarcomatous stroma at the time of first treatment are logically classed with osteogenic sarcomata and bear no relationship to giant-cell tumors.

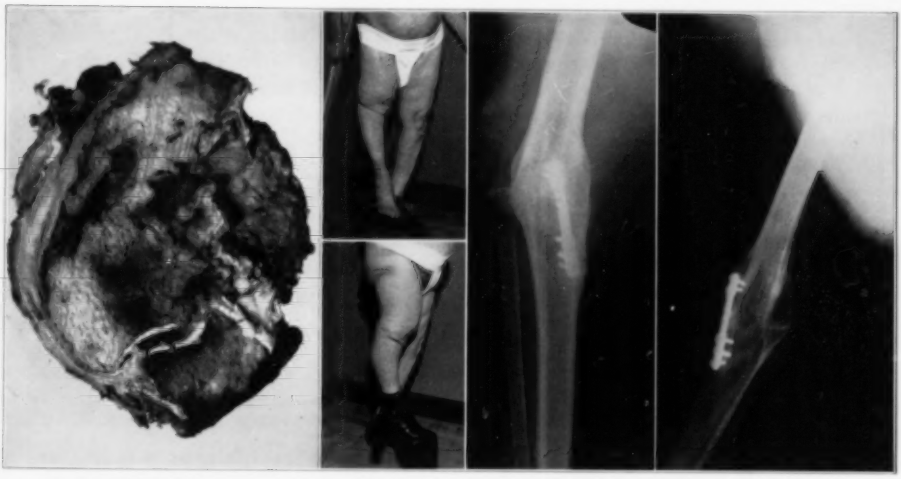

FIG. 4-A FIG. 4-B FIG. 4-C FIG. 4-D

Fig. 4-A through 4-D: Giant-cell tumor of the lower part of the femur in a woman, twenty-nine years old. Wide excision of the tumor was carried out in 1910 after a biopsy showed fibrosarcomatous change.

Fig. 4-A: Surgical portion of the lower part of the femur and upper part of the tibia. (The patient was alive and well in April 1958.)

Fig. 4-B: Shortening of the right lower extremity is shown.

Fig. 4-C: The anteroposterior roentgenogram shows solid fusion.

Fig. 4-D: Lateral roentgenogram, forty-eight years after excision of the tumor.

COMMENT

The longer we follow our cases of giant-cell tumor the higher the rates of recurrence and malignant transformation become. Obviously, conclusions regarding the efficiency of therapeutic methods based on series with short follow-up periods are invalid. Jaffe [6] described a case in which sarcoma developed at the site of a giant-cell tumor twenty-three years after therapy that included irradiation. The present series included one in which the latent interval was fourteen years. It is evident that follow-up of those patients who have received radiation therapy should never cease, and that success rates for those treated with curettage should not be computed on follow-up data of less than ten years. Furthermore, the more assiduously one excludes variants of giant-cell tumor, the more dismal will be the long-term results.

From a review of the present series it is evident that curettage is not the ideal treatment for giant-cell tumor. In 29.6 per cent of patients treated by this method recurrences developed within the first year after therapy. At the end of five years the percentage with recurrences had climbed to 54.1 per cent. In addition to this high rate of recurrence, approximately 10 per cent of giant-cell tumors become frankly malignant and a very occasional one produces metastasis even though histologically benign.

Radiation therapy alone was not successful in controlling any of the giant-cell tumors in our group. There was also no evidence that it diminished the chance of recurrence when used as an ancillary measure with curettage, since the recurrence rate of tumors treated by curettage alone and by curettage combined with radiation was the same. Our experience, as does that of others [1,4,10], indicates that radiation therapy may be influential in the genesis of sarcoma secondary to giant-cell tumor. We believe that it should be employed only for those giant-cell tumors so situated that adequate surgical treatment is impossible.

Although they were only a small part of this total series, those tumors for which complete excision along with removal of all surrounding shells of bone and

fibrous tissue was attempted are an important group from the standpoint of successful treatment (Figs. 3-A to 4-D). If the giant-cell tumors situated in the sacrum, spinal column, ilium, lower part of the tibia, and the skull are excluded, 79 per cent of those in the series—that is, 92 tumors—could have been excised. To be sure, this implies an aggressive attitude in respect to surgery and a willingness to replace the removed bone with a bone graft or a prosthesis, as has been advocated by Kraft and Levinthal for the lower part of the femur. Johnson and Lyford emphasized that this may not be necessary in some instances of excision. Certainly, early excision, when the lesion is small, involves less loss of bone and easier replacement, whether by graft or prosthesis, than excision used as a secondary form of treatment. Other authors[3,13] have shown, and our observations confirm, that excision is the one successful treatment for these tumors.

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DISCUSSION

Dr. Philip D. Wilson, New York, N. Y.: Dr. Johnson and Dr. Dahlin have reported a large series of cases which they seem to have followed long enough for their conclusions to be valid.

Our own series includes thirty-nine additional cases. Unfortunately, from the standpoint of an interesting discussion, I am in such complete agreement with their conclusions that there is little room for debate. Under the circumstances, the best I can do is to emphasize some of the points that they have made.

Because the pathological diagnosis of the nature of the lesion is all important from the standpoint of the treatment and because the pathologist has a hard enough time in determining this with a whole block of tissue in front of him, I think we should always try to supply him with plenty of tissue from biopsy when possible and not ask him to make a diagnosis from tissue removed through a needle.

Furthermore, I do not believe that the clinician should look for too much help from the pathologist in differentiating between aggressive and non-aggressive types of benign giant-cell tumors. The clinician must still accept the responsibility of determining the plan of treatment. He must proceed knowing that from 10 to 15 per cent of these tumors are either malignant, metastasizing tumors at the outset or will become so during the course of treatment.

Whether or not radiation therapy causes malignant transformation remains a matter for

(*Continued on page 947*)

Chronic Neurological Sequelae of Acute Trauma to the Spine and Spinal Cord

Part III

The Syndrome of Chronic Injury to the Cervical Spinal Cord in the Region of the Central Canal

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There are two types of injuries to the cervical spinal cord in the region of the central canal that may be classified as acute or chronic. Although this paper describes the signs and symptoms comprising the syndrome of chronic injury to the cervical spinal cord in the region of the central canal, the pattern of the acute phase must be reviewed to understand the mechanisms and manifestations of the chronic one.

THE SYNDROME OF ACUTE INJURY

For many years there was confusion about the mechanism of injuries of the cervical spinal cord that occurred despite lack of roentgenographic evidence of a fracture or dislocation of the spine. Holmes believed that since the spinal cord was bathed in cerebrospinal fluid it could oscillate with trauma to the vertebral column. Since oscillation of the spinal cord would become asynchronous with that of the osseous spine, the cord would eventually impinge on the spine with resultant contusion of the cord. On the other hand, Cramer and McGowan thought that in acute injuries the nucleus pulposus suddenly protruded, struck the spinal cord, and then recoiled into its interspace, and thus no telltale abnormality was seen in the roentgenogram of the cervical spine. It remained for Taylor and Blackwood and Taylor to shed light on this problem. In 1951 Taylor performed cervical myelography on middle-aged cadavera and found no indentation of the pantopaque column when the cervical spine was in the neutral and flexed positions. However, when the cervical spine was hyperextended there was a defect noted posteriorly in the column of the contrast medium at several of the mid-cervical interspaces. This disappeared when the cervical spine was again placed in the neutral position; Taylor attributed the abnormality to the inward wrinkling of the ligamentum flavum. When a sixty-two-year-old cadaver with hypertrophic arthritic spurring of the cervical spine was examined and the same myelographic studies were made with the cervical spine in hyperextension, there was simultaneous encroachment on the column of the contrast medium anteriorly by the hypertrophic vertebral-body spur and posteriorly by the ligamentum flavum. Taylor believed that the pinching of the cord between these two structures might be the mechanism responsible for neurological disability without any visible anatomical distortion of the cervical spine. Several months after the publication of Taylor's work, a patient was admitted to the hospital for treatment. The history is as follows:

Case 1 *: H. R., a man, sixty-five years old, with hypertrophic arthritic spurring of the cervical spine, was admitted to University Hospital after having tripped on a rug and fallen directly forward on his face. When he regained consciousness he discovered that he was numb and paralyzed in all four extremities. Most of his numbness vanished after a period

* Case 1 was reported in more detail by Schneider, R. C.; Cherry, Glenn; and Pantek, Henry: J. Neurosurg., 11: 546-577, 1954.

of four days, and he regained some motor power in both lower extremities but much less movement in both upper extremities. He had been unable to void and had to be catheterized. On examination, the motion and position senses were intact, and vibratory sensation was spottily preserved, being absent over the lower but present over the upper extremities. There was a level of hypalgesia and hypaesthesia to the seventh cervical dermatome bilaterally and a thermanaesthesia level to the first thoracic dermatome bilaterally. Paresis was present in both lower extremities but was much more marked in all the movements of the upper extremities. There were extensor plantar reflexes.

Roentgenograms of the cervical spine revealed no evidence of fracture-dislocation but merely demonstrated marked hypertrophic spurs projecting posteriorly into the spinal canal at the fourth to fifth cervical interspace.

Cervical laminectomy was performed, with the removal of the laminae of the fourth, fifth, and sixth cervical vertebrae. The denticulate ligaments were sectioned after the dura was opened, and moderate arachnoiditis found at the level of the fourth cervical vertebra. Hypertrophic arthritic spurs were demonstrated at all of the exposed interspaces. Above the most prominent spur at the fourth to fifth cervical interspace there was a noticeable constriction of the spinal cord. Postoperatively, the patient had complete tetraplegia and subsequently died in a nursing home.

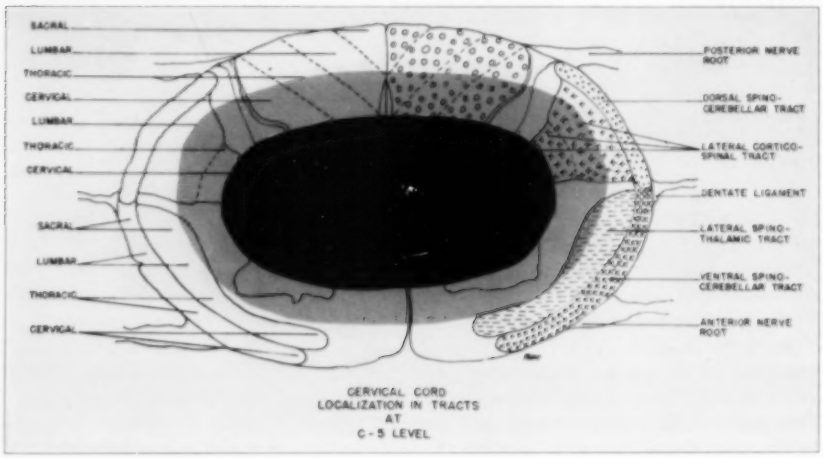

FIG. 1

A cross section of the fifth cervical segment of the spinal cord, illustrating the major sensory and motor tracts and the segmentation within them. The black zone of destruction is surrounded by a gray one of oedema. As the swelling subsides there will be symptomatic improvement so that permanent damage will occur in the motor function of the hands and fingers because these tracts lie within the black zone. (This is a modification of the figure from "The Syndrome of Acute Central Cervical Spinal Cord Injury. With Special Reference to the Mechanisms Involved in Hyperextension Injuries of the Cervical Spine." By R. C. Schneider; Glenn Cherry; and Henry Pantek. J. Neurosurg., **11:** 546-577, Springfield, Illinois, Charles C. Thomas, 1954.)

Comment

There can be no doubt that further injury to the cord resulted from the operation. It was postulated that in the original injury the cervical spinal cord was squeezed—the process described by Taylor being the cause. The resulting compression caused by the wrinkled ligamentum flavum posteriorly and the hypertrophic spur anteriorly was thought to have been concentrated at the center of the cord [21]. This process would be expected to produce the most severe damage centrally in the region of the central canal with damage of decreasing severity to the more peripheral parts of the cord. The medial portions of the lateral corticospinal tracts containing the fibers distributed to the neck, arms, and fingers would, therefore, be more severely damaged than the more peripheral fibers in this tract that are distributed to the bladder, the trunk, and the lower extremities (Fig. 1).

In the event that the cervical spinal cord was not permanently damaged—

there being merely oedema—the patient would be expected to recover spontaneously, with the return of function in the lower extremities first, followed by the return of function in the bladder, arm, and fingers in that order; this we have designated as a syndrome of acute injury to the cervical spinal cord in the region of the central canal.

The patient reported here had shown some recovery of function in the lower extremities. At operation it is presumed that the cord, already damaged in its central portion by the original injury and possibly made more vulnerable by delayed softening, was further compromised by pressure on its lateral aspects in the region of the lateral corticospinal tracts at the time when the denticulate ligaments were being sectioned. In any event, the motor fibers to the lower extremities, which were functional prior to surgery, were irreparably damaged. Because these fibers are situated closest to the lateral aspect of the cord they would be particularly vulnerable.

The prognosis for patients with this syndrome is relatively good. The degree of recovery, of course, is directly proportional to the amount of damage centrally where the compression of the cord is concentrated. Recovery may range from complete or partial return of function to total paralysis with ascending hematomyelia and death.

At the time of the first publication [21], nine cases of the syndrome of acute injury of the cervical spinal cord in the region of the central canal were reported without post-mortem evidence to confirm the clinical thesis presented. In 1958 a second paper [24] was published which described another twelve cases of the syndrome, and this contained the pathological evidence confirming the initial assumption that had been made. A brief résumé of this key case follows:

FIG. 2-A

Case 2. Macroscopically, the cervical spinal cord seemed uninjured. The three transverse incisions indicate the sites at which specimens were procured for microscopic study. (Reprinted by permission from "The Syndrome of Acute Central Cervical Spinal Cord Injury." By R. C. Schneider; J. M. Thompson; and J. Bebin. J. Neurol. Neurosurg. and Psychiat., **21**: 216-227, 1958.)

CASE 2*. On October 11, 1955, J.H., a man, seventy-two years old, sustained an acute injury to the central cervical spinal cord. He had begun to recover in the characteristic manner just described when he suddenly died on November 9, 1955. At post-mortem examination the central-cord damage, which had been postulated originally as being the site responsible for the syndrome, was visualized in the pathological specimen (Figs. 2-A, 2-B, and 2-C).

It should be pointed out that the acute injury to the cervical spinal cord in the region of the central canal may occur in hyperextension injuries in persons with hypertrophic cervical spines, in some cervical fracture-dislocations, and in certain types of cervical vertebral-body compression fractures. In those instances in which the syndrome is due to hyperextension of a cervical spine with hypertrophic arthritis it should be noted that squeezing of the cervical spinal cord may occur at more than one neural segment (Fig. 3).

* Case 2 was presented in detail by Schneider, R. C.; Thompson, J. M.; and Bebin, J.: J. Neurol. Neurosurg. and Psychiat., **21**: 216-227, 1958.

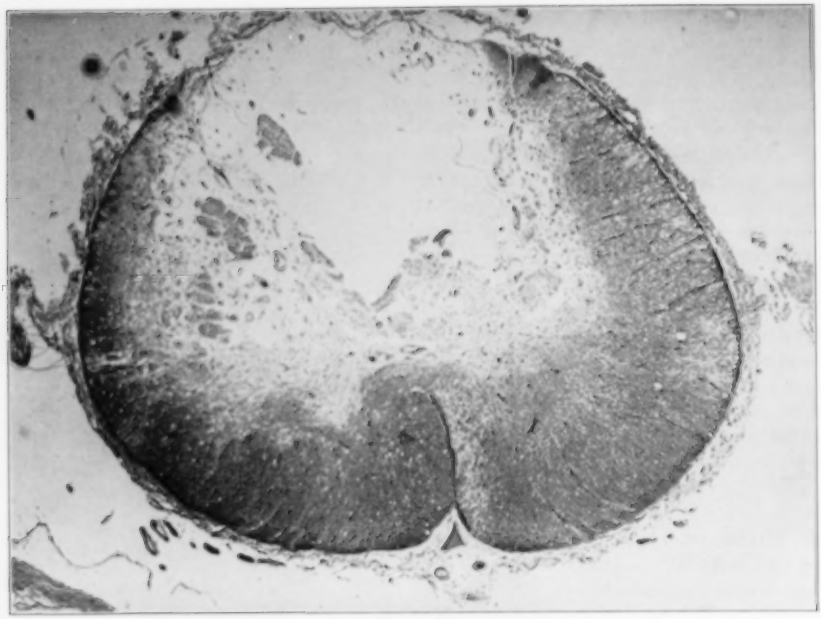

FIG. 2-B

This cross section of the upper portion of the fifth cervical segment of the spinal cord demonstrates an almost complete destruction of the central canal of the cord. The outer or lumbar fibers of the lateral corticospinal tract are preserved, thus permitting movement of the lower extremities. (Reprinted by permission from "The Syndrome of Acute Central Cervical Spinal Cord Injury." By R. C. Schneider; J. M. Thompson; and J. Bebin. J. Neurol. Neurosurg. and Psychiat., **21**: 216-227, 1958.)

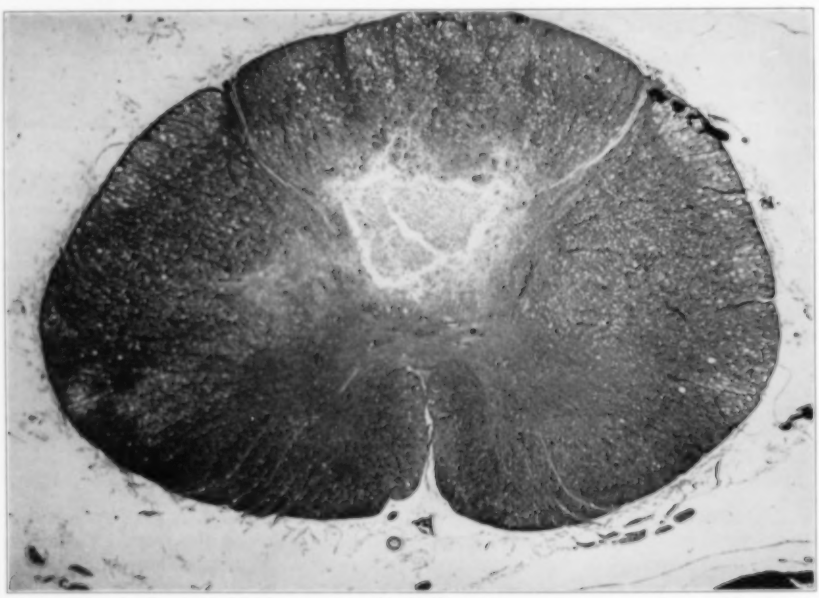

FIG. 2-C

This cross section of the lower portion of the fifth cervical segment of the spinal cord shows merely destruction of the central canal. (Reprinted by permission from "The Syndrome of Acute Central Cervical Spinal Cord Injury." By R. C. Schneider; J. M. Thompson; and J. Bebin. J. Neurol. Neurosurg. and Psychiat., **21**: 216-227, 1958.)

CASE 3 †. G.S., a man forty-nine years old who had had rheumatoid spondylitis involving the cervical spine for ten years, retired at 1:00 A.M. on October 17, 1951, after drinking a large quantity of beer. A few hours later he got up and fell, head first, down a full flight of stairs, landing on his head. After regaining consciousness a few minutes later he could move only his upper extremities, and shortly thereafter he became tetraplegic.

On admission to University Hospital he was well oriented and cooperative. The spine was rigid, and the neck was locked in hyperextension. When the patient was supine his head remained several inches above the examining table. There was marked tenderness over the fourth cervical spinous process. All four extremities were paralyzed, and there was loss of all modalities of sensation up to the fourth cervical dermatome on the right and the

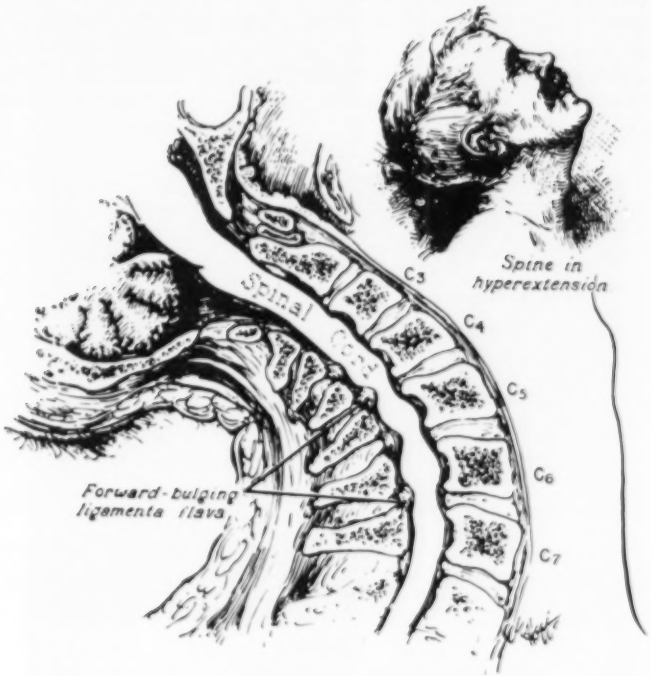

FIG. 3

Diagram illustrating the impingement of the ligamentum flavum on the posterior aspect of the spinal cord. Anteriorly, counterpressure is exerted by the hypertrophic spurs on the vertebral bodies. (Reprinted by permission from "The Syndrome of Acute Central Cervical Spinal Cord Injury. With Special Reference to the Mechanisms Involved in Hyperextension Injuries of Cervical Spine." By R. C. Schneider; Glenn Cherry; and Henry Pantek. J. Neurosurg., **11**: 546-577, Springfield, Illinois, Charles C. Thomas, 1954.)

fifth cervical dermatome on the left. The right triceps and radioperiosteal reflexes were intact. All other reflexes were absent, and there were no pathological reflexes.

Roentgenograms showed changes characteristic of rheumatoid spondylitis, with a comminuted fracture of the lamina and pedicles of the fourth cervical vertebra and an anterior dislocation of the fourth on the fifth cervical vertebra (Fig. 4-A).

Crutchfield skeletal tong traction was applied after placing the patient on a Stryker frame. The intercostal muscles were paralyzed at the time of admission. Diaphragmatic respirations gradually became more labored, and the patient died at 4:00 A.M. on October 20, 1951.

At autopsy the fracture of the fourth cervical vertebra was visualized. The posterior longitudinal ligament was ruptured and fragmented at the fourth to fifth cervical interspace, and the ligamentum flavum was completely torn. The ligamentous structures were not ossified, and the flaval ligaments were of a rubbery consistency; with hyperextension of the neck they might well have bulged into the spinal canal. At the site of the fracture-dislocation the cervical cord was crushed. In addition, for a distance of several segments above

† Case 3 was presented in detail by Schneider, R. C.; Cherry, Glenn; and Pantek, Henry: J. Neurosurg., **11**: 546-577, 1954.

and below the site of injury there was central destruction of the cord (Figs. 4-B and 4-C).

Comment

The roentgenograms demonstrated clearly that the injury had occurred in hyperextension, resulting in marked narrowing of the spinal canal at the fourth to fifth cervical interspace.

The fracture-dislocation presumably caused both anterior and posterior impingement on the cord by the posterior superior margin of the body of the fifth cervical vertebra anteriorly and the laminae and the adjacent flaval ligaments of the third and fourth cervical vertebrae posteriorly. There was complete pulping of the spinal cord with central-canal extension of damage, both cephalad and caudad, over a distance of seven or eight cord segments. With cephalad extension, the residual, functioning diaphragmatic neural segments were compressed, and death occurred from respiratory failure.

FIG. 4-A

Case 3. Lateral roentgenogram, showing a severely comminuted hyperextension injury of the cervical spine with fracture-dislocation of the fourth cervical vertebral body anteriorly on the fifth cervical vertebral body (retouched). (Reprinted by permission from "The Syndrome of Acute Central Cervical Spinal Cord Injury, With Special Reference to the Mechanism Involved in Hyperextension Injuries of Cervical Spine." By R. C. Schneider; Glenn Cherry; and Henry Pantek. J. Neurosurg., **11**: 546-577, Springfield, Illinois, Charles C. Thomas, 1954.)

This case represents an extreme example of central-cord damage, and demonstrates that the lesion in the cord is not necessarily a single continuous one that tapers off in both a caudad and cephalad direction. In this patient's cervical cord the second largest central-cord lesion was approximately six neural segments from the site of greatest damage. There was considerable evidence to suggest that this most distal lesion was a zone of infarction that was caused by a gradient of vascular insufficiency in the anterior spinal artery, with subsequent hypoxia in this segment of the spinal cord. A discussion of this problem is too complex to be described here but may be found in detail in another article [25].

Thus, in some instances, where the injury to the cervical spinal cord is not so severe there may be several isolated central-canal lesions—some due to the local trauma and others due to vascular insufficiency at a level remote from the site of injury. These areas of necrosis may degenerate into cystic lesions and unite to form a single cystic cavity under the influence of the continuous pulsations in the

FIG. 4-B

FIG. 4-C

Fig. 4-B: Autopsy specimen, exhibiting the markedly constricted spinal cord at the fourth to fifth cervical interspace. (Reprinted with permission from "The Syndrome of Acute Central Cervical Spinal Cord Injury. With Special Reference to the Mechanisms Involved in Hyperextension Injuries of Cervical Spine." By R. C. Schneider; Glenn Cherry; and Henry Pantek. J. Neurosurg., **11**: 546-577, Springfield, Illinois, Charles C. Thomas, 1954.)

Fig. 4-C: Multiple transverse sections of the spinal cord, with the uppermost one at the top of the specimen and the lowermost fully six neural segments below the site of constriction. Destruction of the central canal may be seen throughout the entire specimen. (Reprinted with permission from "The Syndrome of Acute Central Cervical Spinal Cord Injury. With Special Reference to the Mechanisms Involved in Hyperextension Injuries of Cervical Spine." By R. C. Schneider; Glenn Cherry; and Henry Pantek. J. Neurosurg., **11**: 546-577, Springfield, Illinois, Charles C. Thomas, 1954.)

cerebrospinal fluid. Parkin, in 1891, reported the post-mortem examination of a patient who had been diagnosed clinically as having post-traumatic hematomyelia. Surprisingly enough, there was not the true syringomyelic cavity that one would anticipate, but merely several areas of necrosis. It was the irregularity in size and the lack of continuity of these acute central-canal lesions that led Frazier, in 1918, to conclude that "hematomyelia uncomplicated and dissociated from any other

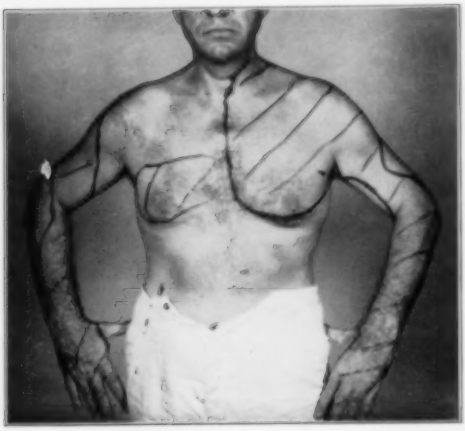

FIG. 5-A

FIG. 5-B

Fig. 5-A: Case 4. The zones of pain and temperature loss are shown.
Fig. 5-B: Lateral roentgenogram showing marked vertebral-body spurring at the fourth to fifth cervical interspace with less deformity at the fifth to sixth cervical interspace.

effect of the injury is not an operable lesion". He did not indicate how such a diagnosis could be made with certainty, but believed that if it could be established myelotomy was not indicated since it did more harm than good. Cohn suggested that it was easy to understand how such a hemorrhagic or necrotic lesion, which was spread over a few segments, might eventually coalesce and form a cystic or syringomyelic cavity. Cohn reviewed the literature on the problem of the late appearance of the symptoms of hematomyelia because of interest in a patient who had fallen from a hay wagon and in whom symptoms of hematomyelia developed eight days later. Cohn discovered that Bramwell, Cassirer, Claude and L'hermitte, Foerster, Heimann-Hatry, Hoch, Kocher, Parkin, Schott, Souques and Henry, Watts, Winkelbauer, Winkler and Jochmann, and Zwirner had reported cases in which there was an interval of a few hours to six months from the time of injury to the onset of the first symptoms of hematomyelia.

THE SYNDROME OF CHRONIC INJURY

There are some patients who have a history of either acute or chronic recurrent trauma in whom progressive weakness develops in the fingers, hands, and arms that is disproportionately greater than the impairment of strength in the lower extremities. The sensory changes vary greatly, but usually take the form of hypalgesia and thermanaesthesia involving the lower cervical and upper thoracic dermatomes. This is the syndrome of chronic injury to the spinal cord in the region of the central canal that has been commonly designated erroneously as syringomyelia.

The following cases illustrate the problems involved.

CASE 4: H. S., a right-handed man, forty-two years old, was first examined at University Hospital on May 18, 1956. He had a history of a two and a half years' loss of temperature sensation and tingling in the outer two fingers of the left hand and the outer side of the left forearm. Within the preceding few months similar right-sided symptoms developed. He also observed that when he cut his neck when shaving there was no pain. Occasionally, there was some dull pain in his neck, and when he coughed or sneezed it accentuated the tingling sensation in his fingers. A slight weakness in the grip developed. Because the roentgenograms showed cervical hypertrophic arthritis and a narrowed intervertebral space he was treated with halter traction, but there was no relief of symptoms.

The patient voluntarily said that five years before (two and one-half years prior to the onset of symptoms) he had been involved in an automobile accident, at which time his car was struck from behind. He sustained a so-called whiplash injury when the head and neck snapped backward sharply into hyperextension, and there was stiffness of the neck for

FIG. 5-C

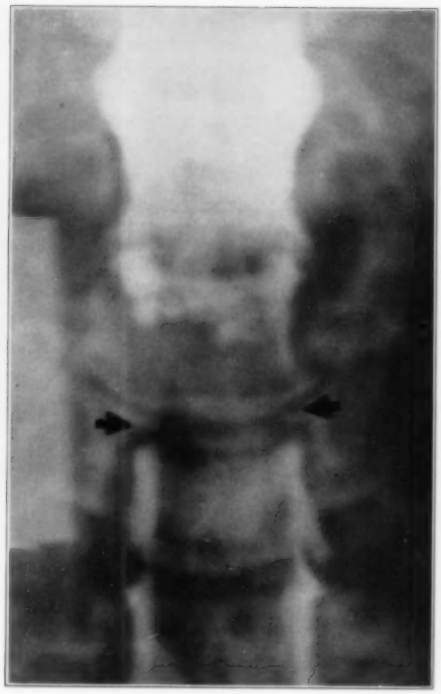

FIG. 5-D

Fig. 5-C: Oblique roentgenogram demonstrating encroachment of the spur at the fourth to fifth cervical interspace upon the intervertebral foramen.

Fig. 5-D. Anteroposterior view of a myelogram showing mid-line filling defect and almost complete block at the fourth to fifth cervical interspace. The head had been tilted upward, and the pantopaque barely passed through the subarachnoid space bilaterally.

about one week. There had been no litigation involved as a result of the accident, and none was contemplated.

On examination there was local tenderness on percussion at the fourth to fifth cervical spinous processes, without any radiating pain. The following positive neurological findings were elicited: The grip of the hand and the extension of the forearm were poor bilaterally. There was atrophy of the left forearm and left hypothenar eminence. The biceps and triceps reflexes were absent bilaterally. He had a definite level of hypalgesia and hypaesthesia from the third cervical to the seventh thoracic dermatome on the left and the fifth cervical to the first thoracic and the fifth to seventh thoracic dermatomes on the right side (Fig. 5-A).

Lateral cervical-spine roentgenograms revealed a marked hypertrophic spurring of the vertebral bodies at the fourth to fifth cervical interspace (Fig. 5-B). This was more graphically shown on the oblique view (Fig. 5-C). On two lumbar punctures the cerebrospinal-fluid pressures, jugular-vein-compression tests, and white-cell counts were within limits. The total protein values were seventy milligrams and seventy-one milligrams per 100 cubic centimeters, respectively. The diagnosis of cervical syringomyelia or cervical intramedullary tumor was made. Because the patient had had two attacks of coronary occlusion and was still functioning well from a neurological standpoint, it was elected to continue conservative therapy until further progression of signs or symptoms developed.

The patient became nervous and impatient, stating that he had had increased neck and radicular pain. He sought further consultation from other neurologists or neurosurgeons, and, finally, on October 6, 1956, Dr. Leo Davidoff, of New York City, ordered a cervical myelogram. The diffuse widening of the pantopaque column was seen at all three of the lower cervical interspaces (Fig. 5-D). Four days later, a cervical laminectomy was performed by Dr. Davidoff at the fourth and fifth cervical levels. On opening the dura, the cord was found to be enlarged, and on incision of it a cystic cavity was demonstrated and drained of a clear fluid. There was no evidence of a neoplasm. A plastic tube was run from this cavity into the subarachnoid space. The denticulate ligaments were sectioned bilaterally, and no attempt was made to remove the hypertrophic spurs on the vertebral bodies. Postoperatively, the patient

had only minimal transient weakness in the left upper extremity and ataxia on finger-to-nose testing. The radicular pain on the right was relieved, and the patient was discharged from the hospital on November 7, 1956.

Comment

In this case the acute injury to the cervical cord in the region of the central canal was presumably caused by hyperextension of the neck in a patient with hypertrophic cervical arthritis, resulting in a sudden, severe squeeze of the cervical spinal cord without fracture or dislocation of the spine. The patient described it as a whiplash injury.

Gay and Abbott first described this lesion, noting that it most often occurred when a halted vehicle was struck from behind, causing the occupant's head to be snapped forcibly backward into sudden hyperextension. In some instances there was a tearing of the ligaments and muscles of the neck, with associated hemorrhage and oedema. In many cases the symptoms of dizziness, stiffness of the neck, increased perspiration, and hyperemotionality did not appear until some days or weeks later. There were relatively few residual neurological signs. Gotten, in reviewing 100 cases of this type of injury, stated that 88 per cent showed recovery after legal claims for damage were completed and over one-half of these patients had no residual complaints. In Gotten's series the patients sixty years of age or more showed a much slower recovery than those in the lower age group, and these older patients believed that they had mild arthritic symptoms possibly related to the injury. Twelve per cent of Gotten's patients still had sufficient symptoms to be receiving further treatment in spite of settlement of all legal claims. There is a possibility that in these patients the symptoms of the syndrome of chronic injury to the cervical spinal cord in the region of the central canal could have developed at a later date. Certainly, this is suggested by the fact that patients who had the slower recovery rate were persons who had arthritic cervical spines or were over sixty years of age. A review of the neurological examinations of this group of Gotten's patients might provide interesting data. Thus, we are very reluctant to use the word *whiplash* because of the medicolegal implications, and therefore refer to these cases as acute hyperextension injuries.

In a recent publication, Brain and Wilkinson reported that roentgenograms of the cervical spine had been made in fifty-two patients with syringomyelia, and in twenty-four patients significant abnormalities of the spine were found. They concluded that the cervical arthropathy which they found in association with syringomyelia was similar to that found in cervical spondylosis, but that the process of degeneration in these patients was intensified by the syringomyelia. These authors also suggested that some of these patients' symptoms might be due to the cervical arthropathy since the neurological symptoms of syringomyelia might be accentuated by cord compression resulting from osteo-arthritic changes in the spine. In one of their six reported cases trauma seemed to have played a part. The location of a head laceration and a concussion suggested the possibility of a hyperextension injury with cord damage in the region of the central canal, but no note was made of any specific correlation between the syringomyelia and the type of trauma.

After a review of Cohn's cases the conclusion could be drawn that our patient (Case 4) had a hyperextension injury that resulted in minor areas of degeneration over one or more segments, which was shown on the myelogram. These segments later coalesced and caused a syndrome similar to chronic syringomyelia. A thorough five-year neurological follow-up of a series of patients similar to those observed by Gotten might yield some interesting data. Certainly, if the relationship between such an acute injury and this chronic disability exists, it is an extremely rare finding in a group of acute hyperextension injuries.

Case 5. B.S., a woman, twenty-nine years of age, was admitted to University Hospital in June 1953 because of paralysis of the fingers, hands, and arms. She was able to walk without support, although she had some weakness in the legs and feet.

FIG. 6-A

FIG. 6 B

Fig. 6-A: Case 5. Lateral roentgenogram showing radically increased lordosis of the cervical spine with projection of the lower part of the fourth cervical body into the spinal canal.

Fig. 6-B: Myelogram showing an almost complete block at the level of the sixth cervical vertebra.

The patient's history was a most unusual and tragic one. In 1941, when she was swinging high in the air, she struck the back of her head on the bough of a tree and was hurled from the swing. She did not recall how she struck the ground, and she was unconscious for a short time. For a period of one week there were severe headache and pain in the neck and one arm, but no other acute sequelae. In 1948 she began to note a gradual, progressive impairment of strength in her hands, fingers, and arms. On several occasions she burned her fingers, and, after the development of the weakness of the upper extremities, there was some spasticity of the neck muscles. By 1949 she began to have impairment of strength in the legs and feet. At her local hospital, lumbar puncture demonstrated no evidence of blockage of the cerebrospinal fluid on the jugular-vein-compression test except when the head and neck were acutely manipulated. The cerebrospinal-fluid findings were negative, and a tentative diagnosis of multiple sclerosis was made. In 1951, after a severe attack of mumps, spasticity of the shoulder-girdle muscles was increased. At this time she began two years of chiropractic treatments. Twice a week during this period she was subjected to a direct, severe blow to the mid-cervical area with a doubled-up or clenched fist. After January 1952, the patient was unable to work or care for herself owing to the rapidity of progression of the symptoms.

At the time of admission to University Hospital in June 1953, there were signs of cranial-nerve abnormalities. There was atrophy of all the muscle groups of the upper extremities but no evidence of fasciculations. There were adduction spasms of both forearms and flexion contractures of the wrists, hands, and fingers. This disability was particularly severe on the right side. The abdominal, back, and lower-extremity muscles were paretic bilaterally. The upper extremities were flaccid but the lower ones were spastic, with bilateral patellar and ankle clonus and bilateral extensor plantar reflexes. Sensory examination showed extreme variations on multiple evaluations. Loss of pain and temperature sensations were demonstrated on the left side over the third cervical to fourth thoracic segments. There was absence of light touch only in the lower extremities, and vibration sense was impaired from the level of the fourth cervical dermatome downward. The status of position sense in the lower extremities was equivocal.

A roentgenogram of the cervical spine showed such a marked lordotic spinal curvature that the first and seventh cervical spinous processes were almost at the extremes of a semicircle (Fig. 6-A). A lumbar puncture revealed a normal cerebrospinal-fluid pressure without evidence of subarachnoid-space block on the jugular-vein-compression test. The total

protein was fifty milligrams per 100 cubic centimeters, and the white-cell count was within normal limits. A cervical myelogram suggested a space-occupying lesion superior to the sixth cervical vertebral level with blockage of the pantopaque at that point (Fig. 6-B).

On June 30 laminectomy was performed from the second through the seventh cervical vertebra.

There was pronounced hyperextension of the spine, with marked angulation of the spinal canal at the fourth cervical level, and associated compression of the spinal cord at this site. Although the dura pulsated above the constriction, no pulsations were visible below this point. The dura was opened in the mid-line, initially preserving the arachnoid. An obvious complete block at the fourth cervical level with swelling of the cord distally for a distance of two segments was revealed. This swelling was responsible for the presence of the apparent space-occupying lesion superior to the sixth cervical vertebral level that had been seen on the myelogram.

The arachnoid was opened laterally, and five pairs of denticulate ligaments were sectioned. When released by this maneuver, the cord seemed to have a normal consistency although its surface had a slightly bluish tinge suggesting contusion or constriction of the fourth cervical segment. The cord was not rotated for fear of causing further respiratory embarrassment. There was no visible evidence of any tumor, and because there was constriction rather than swelling of the cord, myelotomy did not seem indicated or justified. The dura was left open, and the remainder of the wound was closed in layers.

Because of increasing respiratory difficulty, a tracheotomy was performed on July 2, 1953. Three weeks after operation the tonus had diminished in the muscles of the upper and lower extremities. It was now possible to put both upper and lower extremities through a full range of passive motion, and the patient was able to flex slightly the fingers of the right hand. The preoperatively elicited adduction spasm of the upper extremities had disappeared. Four weeks after operation Crutchfield tongs were inserted, and gradually increasing increments of weight added in the hope that the cervical spine might be straightened to some degree. However, since this was not achieved after several weeks, traction was discontinued. The patient was discharged with moderate clinical improvement in the upper extremities. The patient was not available for follow-up examination.

Comment

This patient had a single episode of trauma to the cervical spine followed later by multiple, severe traumatic onslaughts which resulted in marked muscle spasm and subluxation in hyperextension at the fourth cervical vertebral level. In this patient the displaced vertebral body, rather than a hypertrophic arthritic spur, was responsible for the anterior compression of the cervical spinal cord. The posterior compression of the cervical spinal cord could have been due to the wrinkled ligamentum flavum. Despite a clinical picture suggesting the pattern of syringomyelia this patient did not have the usually widened cystic cord for which myelotomy would be performed. Instead, the picture at the operating table was that of severe cord compression.

Case 6. J.B., a retired office worker and farmer, forty-nine years of age, was admitted to the Henry Ford Hospital on May 11, 1958, for investigation of pain in both arms and progressive weakness of the left arm and both lower extremities. The symptoms were of eighteen months' duration.

In 1930, at the age of twenty-one years, he sustained a neck injury in a diving accident and was tetraplegic for a period of two or three weeks, with gradual but almost complete recovery except for minor weakness of the left arm and hand. In the interval between recovery and the onset of his complaints he led an active life as a farmer and also excelled at skiing and ice-skating.

He first noted difficulty with the lower extremities while skiing; he fell more frequently than formerly and it was more difficult for him to get back on his feet after a fall. He noticed a progressive loss of strength in both shoulders, with even a more marked impairment of power in the left hand.

Examination revealed a healthy-appearing man with good muscle development except for obvious atrophy of both shoulder girdles, most marked in the deltoid and supraspinatus muscles and to a lesser degree in the left triceps and interossei muscles. Fasciculations were noted in both deltoid regions but none in the tongue. There was moderate weakness of both deltoids and less of the left triceps and small muscles of the hand. The biceps reflexes were absent, and the triceps reflexes were intact, except that the left one was less active than the right. The reflexes in both lower extremities were hyperactive but there was no clonus. No pathological reflexes were elicited. There was loss of strength in the lower extremities and an impairment of pinprick and light touch over the trunk, extending from the lower portion of the neck to the mid-thorax; this was more pronounced on the left than on the right side. He

had some alteration of disturbance to heat and cold but not a clear-cut loss of these sensations. Vibration and position senses in the lower extremities were intact. He had no gross impairment of sensation of a peripheral type but stated that objects did not feel normal to him in the left hand.

Roentgenograms of the cervical spine revealed evidence of a severe, old injury. This consisted of marked anterior wedging of the third and fourth cervical vertebrae with marked reversal of the cervical curve centering at the fourth cervical vertebra amounting to a rather sharp angulation. The fifth cervical vertebra was displaced posteriorly on the sixth, and the third, fourth, fifth, and sixth vertebrae showed marked degenerative changes with proliferation and encroachment on the fifth cervical intervertebral foramina (Fig. 7).

A myelogram revealed a partial block at the fourth cervical vertebral level, with only a small amount of oil trickling by on either side.

The clinical impression was that this patient had evidence of involvement of the anterior horn cell or nerve root at the level of the fifth and sixth cervical vertebrae and intramedullary changes extending from approximately the fourth cervical vertebra through the upper thoracic region, most likely due to spinal-cord compression or compromised circulation at the site of this previous injury; the former was more likely, as there was evidence of damage to the central canal.

Fig. 7

Fig. 7: Case 7. Lateral roentgenogram of the cervical spine, revealing the evidence of a severe old injury. There is marked anterior wedging of the third and fourth cervical vertebrae with marked reversal of the cervical curve centering at the fourth cervical vertebra with sharp angulation. The fifth cervical vertebral body was displaced posteriorly on the sixth one. The third, fourth, fifth, and sixth cervical vertebrae show marked degenerative change with proliferation and encroachment on the fifth cervical intervertebral foramina.

Comment

The symptoms and signs of chronic injury to the cervical spinal cord in the region of the central canal that were observed in this case were probably on the basis of chronic flexion fracture-dislocation of the cervical spine. It will be interesting to observe whether the signs and symptoms will be corrected merely by laminectomy alone. This should release the posterior-cord-compression factor responsible for squeezing the spinal cord against the angulated vertebral bodies. It is amazing that this patient was able to get along so satisfactorily for a period of about twenty-six years before these disabling symptoms developed. In this case, the proper treatment of the cervical fracture-dislocation by an adequate cervical-spine fusion might very well have prevented the chronic neurological disability.

DISCUSSION

The patients reported in Cases 4, 5, and 6 all demonstrated a syndrome of chronic injury to the cervical spinal cord in the region of the central canal (the chronic phase of acute injury to the cervical spinal cord in the region of the central canal). It is characterized by greater weakness in the fingers, hands, and arms than in the lower extremities. The sensory changes in such patients are extremely vari-

able. It is a counterpart of the syndrome of acute injury to the cervical spinal cord in the region of the central canal, but, in contrast, the patients who sustain the chronic lesion do not have a progressive improvement in pattern. For example, in the acute phase of injury, surgical intervention is contra-indicated, for recovery occurs spontaneously. However, in the syndrome of chronic injury to the cervical spinal cord in the region of the central canal, operation may be indicated in the form of laminectomy with myelotomy (Case 4) or without myelotomy (Case 5) or perhaps simple laminectomy may suffice temporarily (Case 6).

It must be noted that there have been definite reasons for designating the neurological pattern just described as the syndrome of chronic injury to the cervical spinal cord in the region of the central canal, which are as follows:

1. Although the symptoms and signs are the same as those ascribed to syringomyelia this term would suggest the presence of a central cavity or syrinx, which need not necessarily exist in chronic trauma. For example, in Case 5, there was merely simultaneous anterior and posterior compression of the cord, with some partial regression of symptoms after simple laminectomy. There was no evidence of a syrinx.

2. The etiology of syringomyelia, as such, may be neoplastic, infectious, congenital, or so-called idiopathic, as well as traumatic. Therefore, designating this neurological pattern as a traumatic syndrome avoids confusion with these terms.

3. The syndrome of acute injury to the anterior cervical spinal cord [19,21] has as its counterpart a chronic injury to the anterior cervical spinal cord [1,2,5,17,28,29]. In spinal-cord pathology, the syndrome of acute injury to the cervical spinal cord in the region of the central canal [21,24] should have as its counterpart a chronic neurological pattern or syndrome which actually best describes the site of the lesion responsible for the findings and yet avoids citing the pathological change, which, obviously, cannot be determined until the pathological specimen has been examined.

The syndrome of chronic injury to the central cervical spinal cord will be detected if one constantly keeps in mind that such lesions exist. Often, the etiology for such neurological changes is obscure because of the interval (often months or years) that has elapsed between the injury and the appearance of the abnormal symptoms or findings. Therefore, a careful history, a rigorous pursuit of information from old records and roentgenograms; repeated, carefully charted neurological evaluations; and good, accurate post-mortem examination in the region of the cervical cord and cervicomedullary junction are imperative if there is to be a better understanding of such lesions.

SUMMARY

Chronic neurological sequelae develop after a single acute or multiple chronic traumatic episodes to the cervical spine and spinal cord.

The syndrome of acute injury to the cervical spinal cord in the region of the central canal has been briefly discussed to present a foundation for the thesis of this paper.

The syndrome of chronic injury to the cervical spinal cord in the region of the central canal was demonstrated to be a definite entity. It is characterized by a history of previous trauma with the tardy onset of increasing weakness in both upper extremities, which is disproportionately greater than that seen in both lower extremities. There is usually an associated loss of pain and temperature sensations in the dermatomes involving the lower cervical and upper thoracic regions. The syndrome was illustrated by three case reports.

The reasons for using this designation rather than the term traumatic syringomyelia are discussed.

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Surgical Treatment of Congenital Dislocation of the Hip in Children

A One-Stage Procedure *

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During the ten years after World War II approximately 1,250 children were treated at the Orthopaedic Clinic of the Medical Academy in Poznań for congenital dislocation of the hip. Of these children 762 required surgical reduction because of their relatively advanced age or because of failure of previous treatment.

As is generally known, each type of congenital dislocation of the hip requires, in surgical treatment, an individual therapeutic approach.

Simple open reduction is usually sufficient to obtain satisfactory results in children with the acetabulum comparatively well shaped and the femoral head small without increased anteversion and without any high upward displacement. When, however, the head is displaced high, reduction is still possible; but once this is accomplished, the pelvifemoral muscles press the femoral head so powerfully against the acetabulum that ulcerations of the cartilage covering the femoral epiphysis may arise. These joints later show restricted motion, symptoms of intra-articular irritation, and poor end results.

The preparation of patients with preoperative traction makes reduction easier, but does not prevent, according to our experience, undue pressure of the

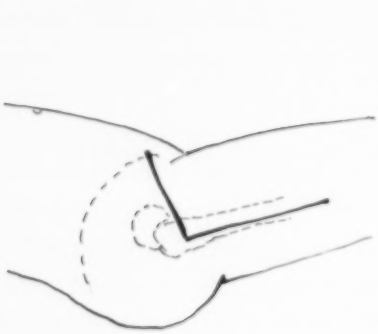

Fig. 1-A

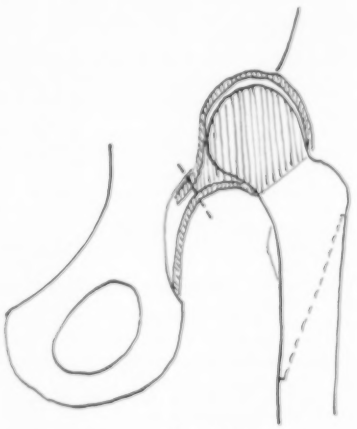

Fig. 1-B

Figs. 1-A through 1-G.: Schematic drawing of the operative technique showing: skin incision (Fig. 1-A); dissection of the articular capsule and its division through the isthmus and the line of the oblique subperiosteal subtrochanteric derotation osteotomy (Fig. 1-B); shortening of the femur (Fig. 1-C); derotation by internal rotation of the proximal fragment on the diaphyseal one (Fig. 1-D); fixation of the fragments after derotation. Two wire loops first penetrate then encircle both fragments (Fig. 1-E); dome-shaped intertrochanteric osteotomy to correct anteversion and valgus deformity (Fig. 1-F); and fixation of the fragments with the aid of an angulated metal plate (Fig. 1-G).

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, New York, N. Y., February 4, 1958.

FIG. 1-C

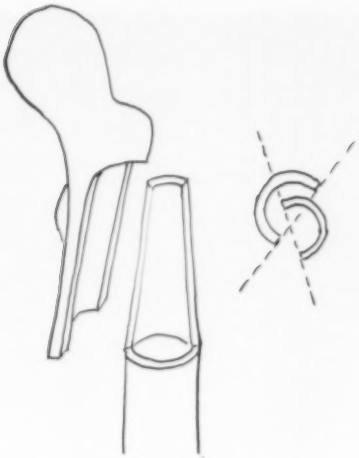

FIG. 1-D

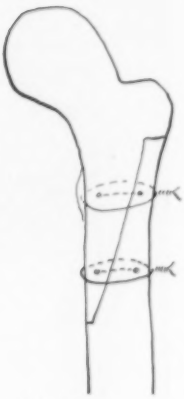

FIG. 1-E

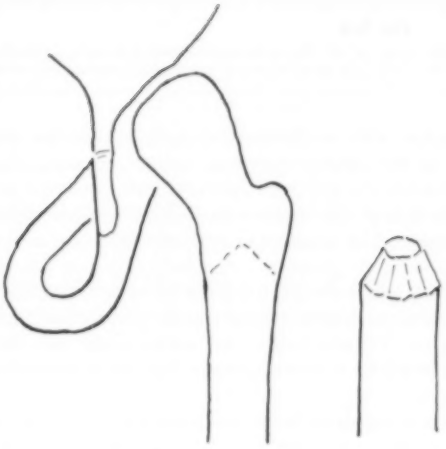

FIG. 1-F

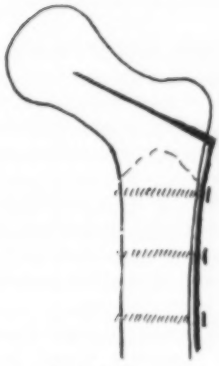

FIG. 1-G

head against the acetabulum. The muscles stretched by extension recover their tone and tension when traction is removed after reduction. The older a child, the more drastic is the problem of muscles shortened by upward displacement of the femoral head.

Camera, Löffler, Ombrédanne [13], and Zahradniček solved this problem by surgical shortening of the femur, thus restoring the balance between the length of muscles, the nerves and artery, and the length of the femur.

Simple open reduction fails if there is markedly increased anteversion of the femoral head and neck. When left uncorrected anteversion is a common cause of poor results following operation. Abnormal anteversion can be corrected by derotational osteotomy of the femur.

Many surgeons perform derotational osteotomy later, at a second-stage operation.

We observed a marked limitation of movements in the knee after supracondylar osteotomy. It usually took a long time to restore the normal range of motion. Supracondylar osteotomy brings, in addition, a danger of slight varus deformity resulting from the pull of adductor muscles.

FIG. 2-A

Roentgenogram made December 18, 1953, of B. R., a five-year-old girl with bilateral congenital dislocation of the hip. The left hip was treated surgically elsewhere, with recurrent actual dislocation.

High subtrochanteric osteotomy with a Küntscher nail to fix the fragments does not prevent rotation of the upper fragment under the pull of external rotator muscles. For this reason Gropič used special nails provided with a sort of fin to prevent relative rotation of the fragments. Zahradniček performed intertrochanteric osteotomy and united the fragments with simple bone nails or screws.

Simple open reduction fails, too, when the acetabulum is constricted by the inverted glenoid labrum, the infolded articular capsule and, when the femoral head is disproportionately large and deformed, and the acetabulum too small, narrow, and shallow to contain the head. In such instances, Colonna performs capsular arthroplasty.

Keeping in mind these facts and considerations we have elaborated a one-stage procedure, based, mainly, on the techniques of Colonna and Zahradniček. We have used this procedure since 1953 in 394 cases of which 172 have been followed up for from two to four years.

OPERATIVE TECHNIQUE

The operative technique is described in Figures 1-A through 1-G.

The skin incision is L-shaped and anterolateral. The hip joint is reached between the sartorius, tensor fasciae latae, and glutaeus muscles. The latter are temporarily detached subperiosteally from the iliac bone. Capsular arthroplasty is performed according to the principles described by Colonna, but without preoperative traction. Thus the patient need not be bedridden before surgery and additional anaesthesia is avoided. Besides this, we bypass the skin infection caused by traction wire, especially in high displacement when great weights are necessary.

The dissection of the capsule from the surrounding muscles should be carried out precisely, especially in the vicinity of the posterior rim and along the iliopsoas muscle. The transverse ligament should be excised completely because otherwise it is difficult to be exact in reducing the head into the acetabulum.

The socket is enlarged and deepened by a hand reamer adapting the depth of the acetabulum to the size of the femoral head, being careful not to make it

FIG. 2-B

Roentgenogram made December 16, 1957, four years after operation on the right side, and two and a half years after the last operation on the left hip. The right hip was graded *A* (clinically good); the left, graded *Co* (clinically poor).

too deep. We deepen the acetabulum only to the point where the triradiate cartilage becomes visible and the acetabulum is hemispheric in shape and large enough to contain the femoral head to a little beyond the mid-line. It is important that the newly formed acetabulum be located and directed in a suitable manner. It is the exposed triradiate cartilage in the floor of the acetabulum which indicates the proper location for reaming. It should be remembered that the normal acetabulum faces forward and downward. If a too high posterior rim of the acetabulum is left, it may act as a fulcrum during external rotation, thus causing gradual redislocation of the head.

In deepening and widening of the acetabulum, care should be taken to avoid pushing small bone particles down beyond the transverse ligament. These particles may give rise to the formation of a bone mass which may limit hip movement.

Acetabuloplasty

Acetabuloplasty is often used, instead of Colonna's arthroplasty, for children three or four years of age. In the young, the socket is frequently deep enough to contain the femoral head, especially when it is small. We simply reduce the head into the acetabulum after excision of the capsule. Sometimes the necessary widening of the socket may be achieved by excision of the infolded limbus. The removal of the transverse ligament is mandatory. Quite often we mold the roof of the acetabulum within the cartilage itself, by cutting away a few layers to form a better support for the head. We always leave the fibrofatty pad on the floor of the socket. We never place the denuded head into the acetabulum when its cartilage is seriously damaged.

Derotation Osteotomy

The derotation osteotomy is an oblique subtrochanteric and subperiosteal procedure. We approach the femur by cutting through the vastus lateralis. An electric saw is used to cut a line from the upper external surface of the femur just below the greater trochanter downward toward the inner surface. By pushing aside the upper fragment, we get a large and easy approach to the acetabulum. Derotation is performed by rotating internally the upper fragment in relation to

FIG. 3-A

Roentgenogram made June 25, 1954, of O. M., a girl, five and one-half years old showing bilateral congenital dislocation of the hip.

FIG. 3-B

Roentgenogram made August 29, 1957, three years after one-stage reduction-reconstruction of the left hip and two and one-half years after the same operation upon the right hip. The roentgenographic result is graded *A*; the clinical result in this case is excellent.

the distal one. When the increased anteversion is combined with a valgus or varus deformity, we use transverse intertrochanteric dome osteotomy. The bone is cut through with an osteotome in such a way that the distal fragment is dome-shaped and convex and the proximal fragment concave; the margin of the concave portion is formed of multiple pedicled osteoperiosteal flaps, which are cut during osteotomy to ensure easy adaptation and large contact of both fragments after osteosynthesis. Additional bone is removed from the distal fragment on the me-

dial or lateral side, depending upon the deformity (valgus or varus), in order to restore the correct angle of the neck and shaft of the femur.

Shortening of the Femur

Shortening of the femur is achieved simultaneously with osteotomy by cutting away a proximal portion of the distal fragment. The bone must be shortened sufficiently to accommodate for the shortening of the muscles. The amount of operative shortening to be done is determined in the following way: after reducing the head into the acetabulum, the fragments are put together and the overriding end of the distal fragment is removed. Surgical shortening usually amounted to from one to three centimeters. In performing intertrochanteric osteotomy we first detach the lesser trochanter and reattach it after the shortening. Derotation is performed by internal rotation of the upper fragment around the distal one. In oblique osteotomy the fragments are fixed by two wire loops which first penetrate and then encircle both fragments. In transverse osteotomy we use a Blount angulated metal plate.

POSTOPERATIVE CARE

After operation, the child is immobilized in a double hip spica. The limb operated upon is kept in slight abduction and neutral rotation.

By the fourth week we remove the anterior portion of the pelvic belt and allow the child to sit in a plaster cast. Two weeks later the cast is completely removed and the patient encouraged to perform active motion.

We do not allow walking earlier than three months following operation; often we prolong this restriction by an additional one to three months. In cases of bilateral open reduction, we perform a second operation after an interval of six months.

Advantages of Method

This one-stage method shortens the period of immobilization to a minimum. There is a great saving of time as compared with the three-stage procedure which requires a longer period of preparatory traction while waiting for the third-stage operation (derotation osteotomy).

COMPLICATIONS

The one-stage procedure is fairly extensive and brings an increased operative risk. Of the 398 children operated upon by this technique since 1953, four died. One child died at the very beginning of the operation, during skin incision; autopsy revealed status thymicolymphaticus. Another child died with symptoms of hyperthermia, but the primary cause of death was, as in the remaining two patients, postoperative shock. But, from the moment we improved our anaesthesia and anti-shock treatment we have had no fatalities among the 208 children operated upon in 1956 and 1957.

Redislocation occurred in seven patients. Six were reoperated upon and the dislocation reduced with three excellent and three fair results. In two children, infection developed and in one there was redislocation. (This patient was mentioned in the group of seven redislocations—the child was not reoperated upon and the result was considered poor.)

In seven patients, late subluxation was noted; the final result was poor. The shaft fragment broke during the operation in two patients, but this incident had no influence on the result of the operation. It is worth mentioning that our Orthopaedic Clinic is a training center for specialists. Therefore, our surgeons change frequently and their skill and degree of training varies widely. Many of them perform this operation for the first time. These facts have, no doubt, a bearing on both the complications and the results.

FIG. 4-A

Roentgenogram made April 19, 1955, of W. A., a girl, four years old, showing congenital dislocation of the left hip, dysplasia with anteversion, and mild vascular changes of the right hip.

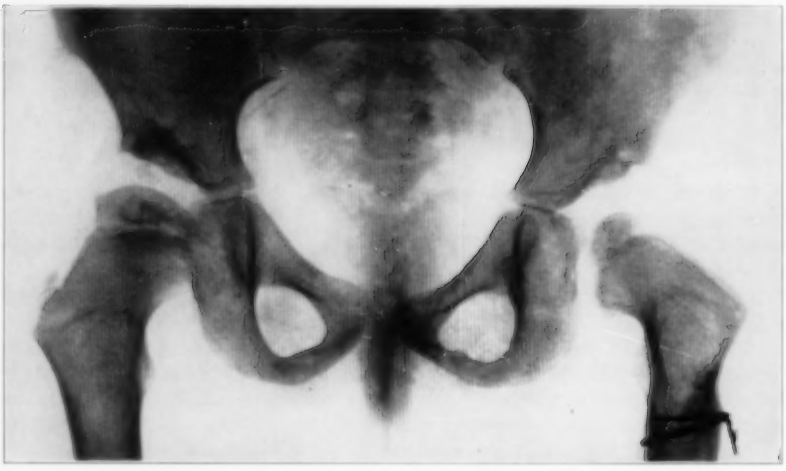

FIG. 4-B

Roentgenogram made September 24, 1955, five months after open reduction of the left hip according to the Zahradníček method.

INDICATIONS

This one-stage procedure for the reduction and reconstruction of the hip is used: (1) as a primary treatment in neglected cases in which treatment is started late, when the patient is between three and seven years of age, and (2) as a secondary operation in cases previously treated unsuccessfully, either by conservative or surgical methods.

The complete procedure is performed when there exists:

1. Great disproportion between the acetabulum and femoral head (a very narrow, shallow, and steep socket or a markedly enlarged and deformed femoral head or both). Capsular arthroplasty is necessary to enable the socket to contain the femoral head and to provide a lasting support for it.

TABLE I

METHOD OF CLINICAL EVALUATION OF RESULTS

	Score *			
	0	1	2	3
I Range of movement (*Degrees*)	Flexion, 0–40; abduction, 0; internal rotation, 0	Flexion, 45–75; abduction, 5–10; internal rotation, 5–10	Flexion, 80–110; abduction, 20–30; internal rotation, 20–30	Flexion, over 110; abduction, normal; internal rotation, normal
II Gait	Marked limp; Trendelenburg, +; Duchenne, +	Slight limp; Trendelenburg, ±; Duchenne, −	Slight limp only after running or long walk	Normal
III Pain	Pain during walking	Pain when walking long distances	No pain	
IV General efficiency	Greatly reduced activity; easily fatigued; does not participate in games	Slightly reduced activity; fatigue only after long walks; participates in games	Normal	

* The total score is obtained by adding the points in columns I, II, III, and IV.

2. High upward displacement of the femoral epiphysis. Shortening of the femoral diaphysis is needed to overcome the shortening of muscles and to prevent undue pressure of the femoral head against the acetabulum after reduction.

3. Anteversion of the femoral head and neck of more than 45 degrees. Derotation is indicated to ensure stable reduction and good congruency of the head and acetabulum.

4. Valgus deformity greater than 145 degrees. This needs to be reduced to improve hip statics and to prevent deterioration and overloading of the femoral head.

FIG. 4-C

Roentgenogram made February 8, 1957, twenty months after operation. The process of adaptation of the acetabulum to the femoral head can be readily seen. This is, however, possible only in children under five years of age. Anteversion has been corrected in the right hip by subtrochanteric oblique osteotomy. The vascular changes disappeared.

TABLE II

ROENTGENOGRAPHIC RESULTS IN RELATION TO AGE

Age (Years)	Results *							
	A	Ao	B	Bo	C	Co	D	Do
3	18		20	2				
4	15	1	19	5		1		
5	9	1	12	7	1	2		
6	7	1	13	8				
7		3	7	3		1		
8	1	1	1	6		2		
9			1	3				
12								1
Total	50	7	73	34	1	6		1

* A: Normal or nearly normal appearance of the hip joint.
B: The femoral head is not quite centered within the acetabulum but not actually subluxated; the acetabulum is sufficiently deep to contain a well shaped head and to provide adequate support for it; the acetabular roof is oblique (less than 30 degrees); the anteversion is less than 45 degrees; and the angle formed by the femoral neck and shaft is from 100 to 145 degrees.
C: Recurrent subluxation.
D: Recurrent dislocation (Fig. 2-A).
o: Vascular changes. Such a condition is denoted by a joint index, for example, Co (Fig. 2-B).

When all of these conditions are not present, we perform only those parts of the operation necessary to correct the existing abnormalities.

RESULTS

The clinical evaluation of the results are expressed in Table I. We assumed a total score of ten points as an excellent result, eight to nine points as good, five to seven points as fair, and less than five points as poor.

The roentgenographic evaluation in relation to age is seen in Table II. The assessment of results expressed by indices is more objective than other methods, although some subjective factors cannot be avoided.

The method of grading results should be simple to be practical. Therefore, we created our own way of estimating results, applying the system of points, somewhat different from that of D'Aubigné and of Massie and Howorth. This was especially justified, since we evaluated the results in children only.

In the clinical assessment of results four factors were taken into account: range of movement, gait (limp), pain, and fatigue (general activity of the child).

When estimating the range of motion after operation three index movements were considered: flexion, abduction, and internal rotation.

Flexion: Flexion is the most significant motion in function of the hip. Its range is usually in proportion to other motions of the hip.

Abduction: Abduction, when present in an adequate range, means that there are no serious disorders within the hip, particularly no vascular change. Vascular change or any other intra-articular irritation brings about, apart from flexion contracture, the tendency to adduction contracture thus decreasing or abolishing abduction. We have never observed an abduction contracture in children we treated since postoperatively the hip is immobilized in a plaster cast in only slight abduction.

Internal rotation: We found after surgical correction of the anteversion that the limb shows a tendency to assume some degree of fixed external rotation. The presence of internal rotation was therefore a hint that this tendency had subsided. (We did not see fixed internal rotation in many of our patients.)

Four classes of the range of motion, giving the score of from zero to three points, were made. They were chiefly related to the range of flexion:

1. When flexion is less than 40 degrees the child cannot sit or perform any activity requiring flexion in the hip. Here the score is zero.

FIG. 5-A

Roentgenogram made October 20, 1955, of G. B., a girl, five years old, showing congenital subluxation of the right hip.

FIG. 5-B

Roentgenogram made March 7, 1957, seventeen months after operation. This case points out that the one-stage reduction-reconstruction can also be successfully performed in subluxations. There is still enough capsule to cover the femoral head completely.

2. Flexion from 45 to 75 degrees enables the child to sit, but due, largely, to motion of the lumbar spine. Lumbar kyphosis occurs during sitting, a considerable strain being imposed upon the spine. The child can also reach his feet with his hands. The score is one point.

3. If flexion is from 80 to 110 degrees, sitting is easy and normal; the child can even sit down on low objects and assume a crouching position. The score is two points.

FIG. 6-A

FIG. 6-B

Fig. 6-A: Roentgenogram made June 21, 1955, two years after one-stage reduction-reconstruction performed for congenital dislocation of the right hip. The right femur is only four millimeters shorter than the left, although it had been shortened three centimeters during operation.

Fig. 6-B: The right tibia is two millimeters longer than its mate. The total shortening of the limb operated upon is only two millimeters. In the majority of our patients, the discrepancy in the limb length caused by operative shortening, ranging from one to three centimeters, disappeared completely within the first two years following operation.

4. When the flexion is more than 110 degrees it may be regarded as practically normal, since all functions can be done readily without placing abnormal strain upon the spine. The score is three points.

Gait was subdivided into four classes:

1. Zero: marked limp, the same or almost the same as before operation; positive Trendelenburg and Duchenne signs;

2. One point: slight limp, much less than before operation; positive Trendelenburg and Duchenne signs;

3. Two points: normal gait on examination, but slight limp appearing in the evening after long walks or while running; and

4. Three points: normal gait under all circumstances.

Pain was evaluated by subdividing it into three classes:

1. If the pain is marked, occurring at each step or frequently, the score is zero.

2. If slight pain appears in the evening, after long walks, or at weather changes, the score is one point.

3. If there is no pain, the score is two points.

Fatigue (general activity) was evaluated in a similar manner to pain:

1. Quick fatigue, which restricted general activity and made only short walks possible, was graded as zero.

TABLE III

CLINICAL RESULTS IN RELATION TO AGE

Age (*Years*)	Results				No. of Patients
	Excellent	Good	Fair	Poor	
3	18	16	5		39
4	20	15	5	2	42
5	11	14	2	3	30
6	10	10	8	3	31
7	4	2	3	5	14
8	1		7	3	11
9			3	1	4
12				1	1
Total	64	57	33	18	172

TABLE IV

CLINICAL RESULTS IN PER CENT OF THREE AGE GROUPS

Age (*Years*)	Results			
	Excellent	Good	Fair	Poor
3 to 5	44	40	11	5
6	32	32	26	10
7 to 12	17	7	43	33

TABLE V

COMPARISON OF CLINICAL AND ROENTGENOGRAPHIC RESULTS

Clinical Result	Roentgenographic Result								
	A	Ao	B	Bo	C	Co	D	Do	Total
Excellent	25	2	35	2					64
Good	23	1	26	7					57
Fair	2	2	8	18	1	2			33
Poor		2	4	7		4		1	18
Total	50	7	73	34	1	6		1	172

2. Fatigue arising in the evening, after long walks, and only slightly restricting the child's activities was scored as one point.

3. No abnormal fatigue and no restriction of activity was scored as two points.

A total score of ten points was assumed as an excellent result: eight to nine points, good; five to seven points, fair; and less than five points, a poor result.

OPERATIVE RESULTS

From 1953 to the end of 1957, 398 hips were operated upon by the method described previously. The 172 hips operated upon from 1953 to 1955 were followed for a period of two to four years. The ages of the children ranged from three to twelve years.

Tables III and IV show the clinical results. There is a striking difference between the group of children less than six years old and of those over seven. In the former group the percentage of satisfactory results (excellent and good) is very high (84 per cent), while in the latter, rather low (23 per cent). Six seems to be the border-line age if satisfactory results are to be expected. At this age favorable results still constitute 64 per cent. The comparison of clinical and roentgenographic results is shown in Table V.

FIG. 7-A

Roentgenogram made January 26, 1953, of K. H., a girl, three and one-half years old, showing congenital dislocation of both hips.

FIG. 7-B

Roentgenogram made May 14, 1957, four years after open reduction, by Zahradníček's method, on the left hip, and nearly four years after one-stage reduction-reconstruction of the right hip. Roentgenographic result is graded *A*, and the clinical result, excellent.

Vascular Changes

Vascular changes of the femoral head were found in 28 per cent of patients operated upon. Massie and Howorth observed these changes in 41 per cent of their patients but Colonna did not state any percentage.

Twenty-eight per cent seems a rather high rate and might be related by some surgeons to the operative method itself. We must therefore explain that we took into account all vascular changes observed after operation.

These changes can be roughly classified into two groups: (1) Slight and transient, subsiding within several months without leaving any bad effect. This group constituted 70 per cent of all vascular changes and occurred mainly in children less than six years old and (2) Severe and persistent, lasting for years. These constituted 30 per cent of all vascular changes and occurred almost exclusively in children operated upon when they were over six years old, or in children having marked vascular changes before operation as a result of previous unsuccessful attempts at reduction performed elsewhere.

In view of these findings we have now decided not to operate on older children by the one-stage method, especially when they have bilateral dislocation. Restriction of this operation to children under six will undoubtedly improve our results as far as vascular changes are concerned.

The assumption that the one-stage procedure, including subtrochanteric osteotomy to reduce valgus deformity and anteversion as an essential step of operation, accounts for vascular changes has very little if anything to support it.

Our knowledge of the vascular anatomy of the femoral head and neck [9,15,16] indicates definitely that osteotomy performed at the subtrochanteric level, as well as careful dissection of the articular capsule and its division through the isthmus or at the acetabular attachment, cannot by themselves produce vascular changes, provided the femoral attachment of the capsule is respected. We avoid disturbing this region as far as possible.

After the one-stage procedure was performed on younger children severe vascular changes were rare.

Subtrochanteric osteotomy to correct valgus or varus deformity when performed as the sole procedure never caused vascular changes; on the contrary, when these changes were present before operation, they seemed to clear up more quickly after osteotomy.

It is impossible at this time to give a final estimate of the results of the impairment of the arterial-blood supply to the femoral head observed in our patients. Moderate and severe changes persist for several years before they are healed [10]. Much longer follow-up is necessary before the final results of this operation will be known.

CONCLUSION

1. The operative procedure referred to in this paper aims at the removal of all abnormalities of the dislocated hip in a one-stage operation, thus shortening to a minimum the time necessary for treatment.

2. The most favorable age for this procedure ranges from three to five years; of 111 children in this age group, ninety-four had excellent and good results, twelve had fair, and only five had poor results.

3. Six is the maximum age limit at which it is still possible to expect a satisfactory result.

4. Whereas the majority of failures in the group of children between the ages of three to five years may be attributed to faulty operative technique, the bulk of failures in children over six are due to vascular changes which develop despite correct operative technique.

6. The follow-up period (two to four years) is too short to allow a definite conclusion to be drawn. This paper must then be regarded as a preliminary report.

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DISCUSSION

Dr. John R. Moore, Philadelphia, Pennsylvania: This is an excellent paper. The operative procedure is based upon the premise that deformity of the acetabulum, femoral torsion, coxa valga and vara show very little if any tendency to spontaneous improvement. One must conclude that the author considers these anomalies as a primary congenital deformity. However, overwhelming evidence—roentgenographic, experimental, and clinical—has shown these abnormalities to be secondary to the dislocation. Successful reduction of the hip during infancy and the proper treatment of the subluxation during infancy offers strong opposition to the premise upon which the surgical procedure is based, entirely or partly. The operative procedure therefore does not have a strong basis of support. The procedure itself is well illustrated. It is quite obvious that the blood supply of the proximal fragment of the femur is dependent almost entirely upon the external rotator flap, the glutaeus medius and minimus attachments to the greater trochanter, and the mild periosteal flap (vasta medialis and lateralis). The blood supply is therefore in considerable jeopardy and aseptic necrosis is a highly probable complication. The vascular changes in 48 per cent of the patients as reported by the author are not surprising. The two to four-year follow-up is probably too short a time to evaluate the end results, and in all probability the number of cases of vascular necrosis will increase and the 96 per cent with excellent results will decrease with time. A five-year follow-up, or longer, will prove or disprove this conjecture. The relatively large number of dislocated hips would indicate the need of more intensive education of the layman and physician in the early recognition of congenital dyplasia and congenital dislocation of the hip. It is regrettable that more emphasis is not placed on this fact.

Lumbosacral Anomalies

BY THEODORE A. WILLIS, M.D., SAN MATEO, CALIFORNIA

Structural variations of the lumbosacral mechanism have been discussed repeatedly, particularly those that involve the transverse processes in various stages of sacralization of the last lumbar vertebra and those that affect the elements of the neural arch, resulting in instability of the spinal column on the sacrum or endangering the nerve roots at their intervertebral exits.

The purpose of this paper is to report the results of a statistical study of certain variations of the lumbosacral joint, a study undertaken preliminary to further investigation of lumbosacral stability. Tht variations studied were (1) the anteroposterior diameters of the last lumbar and first sacral vertebral bodies, (2) the lengths of the pedicles as they would be visualized in lateral roentgenograms, (3) the anteroposterior inclination of the joint surfaces of the sacral articular processes with respect to the superior surface of the sacrum, and (4) the angles formed by the planes of the joint surfaces of the posterior articulations with the sagittal plane. The statistics are based on a recent examination of 100 skeletons chosen at random from the Todd Collection of Western Reserve University School of Medicine. Skeletons showing partial sacralization or defects of the neural arch were excluded.

BODY DEPTH

A number of years ago, I discussed the often greater depth of the last lumbar body as compared with that of the adjacent first sacral vertebra, which, in lateral

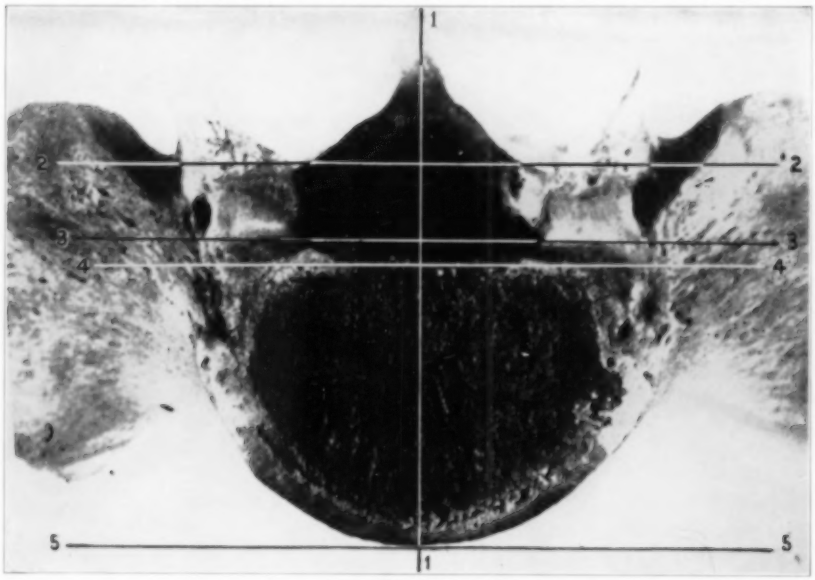

FIG. 1

The superior surface of a sacrum having a *concave* posterior body surface. Line 1, the sagittal plane. Line 2, the most anterior points of the superior articular surfaces. Line 3, the points of greatest body depth. Line 4, greatest depth of the posterior body concavity. Line 5, the anterior border of the centrum. Body depth is measured from Line 5 to Line 3, pedicle length from Line 3 to Line 2, and depth of the concavity from Line 3 to Line 4.

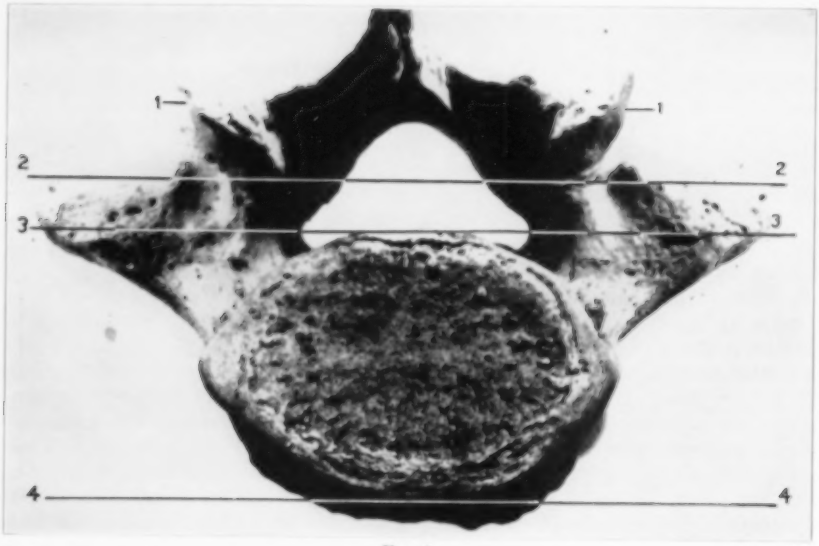

FIG. 2

The inferior surface of a last lumbar vertebra. Line 1, inferior articular facets, asymmetrical and convex. Line 2, the most anterior points of the facets. Line 3, the posterior border of the centrum. Line 4, the anterior border. Body depth, Line 4 to Line 3; pedicle length, Line 3 to Line 2.

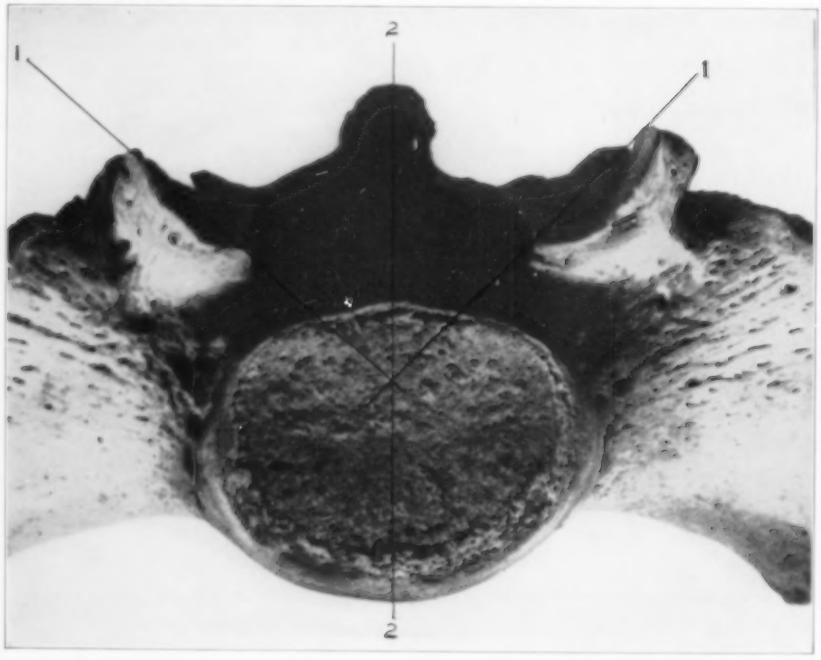

FIG. 3

The superior surface of a sacrum with a *convex* posterior body surface. Note that the articular facets are asymmetrical in form but are at the same angulation with the sagittal plane, as shown by Line 1 and Line 2.

roentgenograms, gave rise to an optical illusion of backward displacement of the former on the latter [3]. I reported then that in measurements of fifty skeletons, the depth of these bodies was equal in only 34 per cent. In the other 66 per cent, lumbar depth exceeded sacral depth or in a few instances was less—which was an incidence six times as great as that which had been reported to be of etiological significance in backache [1,2].

In both the earlier and the present series of skeletons, the depth of the segments was measured from the most anterior to the most posterior parts of the bodies as they would appear in true lateral roentgenograms, rather than at their midlines, because, in about 25 per cent of each series, the posterior surfaces of the first sacral bodies were concave—a condition not discernible by roentgenograms (Figs. 1 and 2).

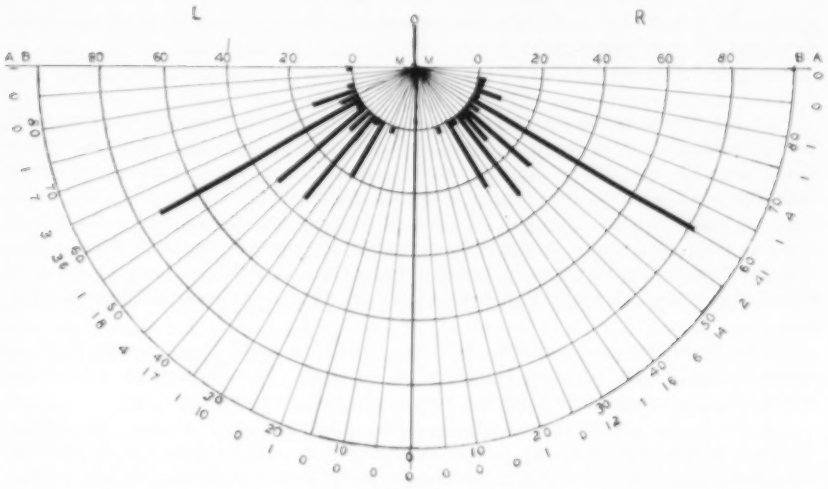

FIG. 4

Chart showing the incidence of the various degrees of angulation of the articular surfaces of the lumbosacral posterior joints. In this chart each facet was given a value of two millimeters and its angulation was recorded on the closest 5-degree radial. The circumferential figures denote: arc *BB*, the degree of angulation, and arc *AA*, the incidence at each radial.

In nineteen of these 100 skeletons, the adjacent last lumbar and first sacral bodies were of equal depth; in twelve, the sacrum was deeper; and in sixty-nine, the lumbar was deeper. The greatest excess of lumbar depth over sacral was ten millimeters; the next greatest was eight, then seven, and, twice, six millimeters.

PEDICLE LENGTH

Pedicle length was considered to be the distance from the most posterior part of the vertebral body to the most anterior part of the articular facets, as these points would appear in lateral roentgenograms (Figs. 1 and 2). The lumbar inferior articular processes are located posterior and superior to those on the sacrum. It was anticipated that the amount of posterior overriding of the lumbar processes with respect to those of the sacrum would depend on this inclination in the coronal plane. It was also anticipated that the length of the lumbar pedicles would increase as this inclination decreased, that is, as the plane of the facets approached the coronal plane. This was not always true. In fact we found no correlation between pedicle length and body depth. In twenty specimens the sacral pedicles were one or two millimeters longer than the lumbar; in one in which there was marked bone lipping about the facets, they were five millimeters longer. In

twenty-five, the lumbar and sacral pedicles were of equal length and the corresponding vertebral bodies were of approximately equal depth. In fifty-five, the lumbar pedicles were longer than the sacral. The greatest excesses were seven, six, and five millimeters in three specimens, and four millimeters in two specimens.

INCLINATION OF FACETS

The superior articular facets of the sacrum have frequently been described as extending downward and slightly backward in relation to its superior surface. To such backward inclination has been attributed posterior displacement of the last lumbar segment on the sacrum when the intervening disc has degenerated with narrowing of the interspace. Only one of these 100 sacrums showed such inclination. In eighty-two, the planes of the facets and of the superior surfaces were at right angles. In seventeen, the facets inclined downward and forward, but seven of these were unilateral, with the opposite facet at right angles to the superior surface of the sacrum. The greatest forward inclination among these seventeen specimens was 10 degrees.

ANGULATION OF FACETS

In cross section the articular surfaces of the sacral articular processes were, with few exceptions, concave, whereas the lumbar articular surfaces were convex. The angles formed by the plane of these articular surfaces (as measured across their concavities) and the sagittal plane of the vertebral bodies (Fig. 3) varied from 20 to 90 degrees. The average was 49 degrees and the mode, on both right and left sides, 60 degrees. A single facet with a 90-degree inclination was diminutive and flat. Inclination was bilaterally asymmetrical in 52 per cent. One was at 80 degrees, two at 75, and the others diminished to a minimum of 20 degrees (Fig. 4).

The frequency and amount of these variations seem to eliminate them as being of clinical importance, except when extreme. The findings in this study, which also are inconsistent with the commonly accepted opinions, are:

1. That the posterior projection of the inferior border of the last lumbar vertebra beyond the opposing border of the sacrum is not indicative of posterior displacement of the lumbar vertebra but usually only a difference in the anteroposterior diameters of the two vertebral bodies;

2. That the planes of the joint surfaces of the superior sacral articular processes do not incline backward in relation to the superior surface of the sacrum;

3. That the planes of the lumbosacral articulations rarely approach either the sagittal or coronal planes.

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Localization of Alkaline Phosphatase in the Histogenesis of Cartilage and Bone*

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The role of alkaline phosphatase in the process of bone formation was first shown by the studies of Robison (1923); subsequently Gomori (1939) published a method for the histochemical localization of alkaline phosphatase. Since that time, various investigators have studied this enzyme in association with ossification, calcification, and dentine deposition. Gould and Shwachman (1942) reported that bone phosphatase and serum phosphatase are elevated in the presence of excessive new-bone formation but that they are diminished in scurvy. Follis (1951) reported complete absence of alkaline phosphatase in the *Gerüstmark* cells in ribs of scorbutic guinea pigs. Gutman and Gutman (1941) suggested that it is the presence of phosphorylase in calcifying cartilage which converts glycogen into hexosephosphate. Robison (1923) suggested that this hexosephosphate is a substrate for alkaline phosphatase. In view of these findings, one's attention is invited to the hypothesis of Gutman and Gutman that, in cartilage, phosphorylase converts glycogen to hexosephosphate, and alkaline phosphatase, a hydrolytic enzyme, then acts on this esterphosphate to liberate a phosphate ion which combines with calcium ions in the calcifying cartilage matrix when the solubility product of the phosphate ions is exceeded. The investigations of Freeman and McLean (1941), Kabat and Furth (1941), Horowitz (1942), Gomori (1943), Bourne (1944), Greep and associates (1948), Follis and Berthrong (1949), and Lorch (1949), among others, have dealt with the association of alkaline phosphatase with calcification and ossification. It is now generally accepted that osteoblasts, odontoblasts and ameloblasts, and hyaline cartilage cells when they become hypertrophic, all manifest alkaline phosphatase activity.

It has not been clearly demonstrated, however, at what stages of mesenchymal cellular differentiation this enzymatic activity appears, nor is it known whether this enzyme is present in both osteogenetic and chondrocytic precursors prior to their differentiation to either osteoblasts or chondroblasts.

To study such a problem in relation to bone development, it is desirable to do so at a site where both membranous and endochondral bone are differentiating simultaneously. Because such features are present in the developing mandible, Meckel's cartilage, the tympanic ring, and the anterior process of the malleus of the developing rat foetus it seemed appropriate to make a detailed study of this area with respect to the presence of alkaline phosphatase in association with different modes of osteogenesis. The histogenesis of these structures in man has been previously described by Richany, Anson, and Bast (1954), Anson, Bast, and Richany (1955) and Richany, Bast, and Anson (1956) and include the following interesting features:

1. The mandible shows varied types of ossification. It begins to ossify classically as a membranous bone; with further fetal growth it acquires a preskeletal type of tissue in the developing upper rami and condyles, the histogenesis of which has not been previously explained. This tissue appears as a "neomorph" intermedi-

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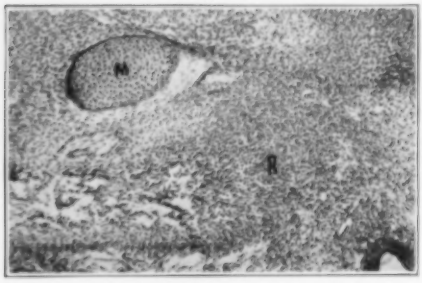

FIG. 1

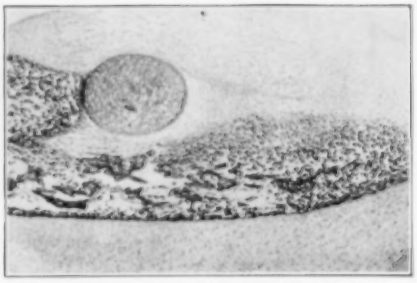

FIG. 2

Fig. 1: Photomicrograph of a rat fetal mandible (fifteen days from first day of pregnancy) stained with hematoxylin and eosin (× 50). Meckel's cartilage (*M*) and the early stages of condensation and differentiation of mesenchymal cells to osteogenetic cells (*R*) are seen.

Fig. 2: Photomicrograph of an adjacent section (× 50) showing alkaline phosphatase activity which is represented by a black precipitate of cobalt sulphide in the nuclei of osteogenetic cells and in some of the formed osteoid matrix. The surrounding mesenchyme is unstained.

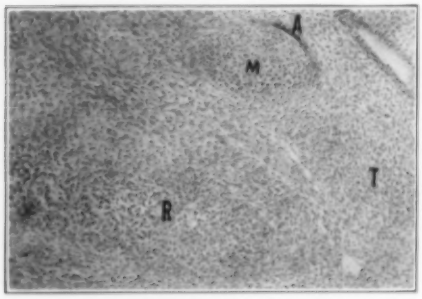

FIG. 3

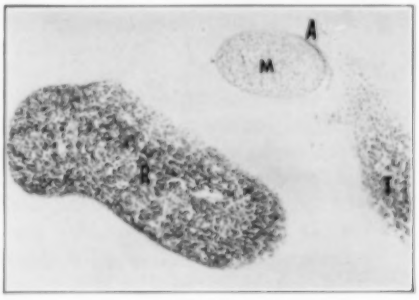

FIG. 4

Fig. 3: Photomicrograph of a section of a rat fetal mandible (sixteen days) stained with hematoxylin and eosin (× 50) shows the anlage of three membranous sites of ossification. The mandible (*R*), tympanic ring (*T*), and the anterior process of the malleus (*A*) in the peripheral perichondrium of Meckel's cartilage at (*M*).

Fig. 4: An adjacent section of same stage stained for alkaline phosphatase which is represented in a black precipitate in the ossification sites that are well demarcated from the general mesenchyme which is unstained. Ramus (*R*), Meckel's cartilage (*M*), anterior process (*A*), and tympanic ring (*T*) are shown (× 50).

ate between cartilage and bone. It is characteristic of this tissue to either transform directly into bone or to develop cartilage and then undergo replacement as in endochondral ossification.

2. Meckel's cartilage is an embryonic transient structure which appears during the development of the temporomandibular joint. The anterior portion ossifies by endochondral bone formation while its mid-portion, to the level of its continuity with the malleus, does not normally ossify but attains a mature chondrocytic texture and then dedifferentiates into a connective-tissue ligament.

3. The tympanic ring and anterior process of the malleus, the latter developing in the perichondrium of Meckel's cartilage, are classically intramembranous bone and retain this mode of osteogenesis into adult form.

The primary object of this study is first to present histological evidence in rats demonstrating the presence of alkaline phosphatase in the normal process of differentiation of embryonic mesenchymal cells to cartilage or bone, and second to determine the role, if any, of alkaline phosphatase in the ossifying anterior portion of Meckel's cartilage, as contrasted to the non-ossifying mid-portion of this structure. Further, the histogenesis of the preskeletal tissue, or secondary cartilage, which appears in an intramembranous site of ossification in the rami and condyles of the mandible may shed some light on the origin of this tissue.

Does this tissue originate from a stem cell with osteogenetic properties and change its pathway of differentiation to give rise to an intermediate type of preskeletal tissue which can either transform to bone or continue to cartilage, or does this tissue derive from embryonic mesenchymal cells that have differentiated solely through the various stages of chondroblastic precursors?

Our description will be limited to rat fetal material.

MATERIALS AND METHODS

This investigation of the alkaline phosphatase activity in the histogenesis of bone and cartilage was made on heads of fetal rats between fifteen to twenty days of intrauterine life. The first day of pregnancy was timed by finding spermatozoa in daily vaginal smears. At the time of sacrifice the petrous region and mandible were removed and fixed simultaneously in the following fixatives: cold acetone, cold absolute alcohol, and isopropyl alcohol. Cold absolute alcohol and cold acetone proved most satisfactory. The tissues were not decalcified. Dehydration was accomplished only by cold absolute alcohol or cold acetone. Cedar-wood oil was used for clearing; subsequently tissues were embedded by three changes of paraffin twenty minutes each at 52 degrees centigrade. Sections were cut serially at eight micra. Alternate adjacent sections were stained with hematoxylin and eosin and for alkaline phosphatase. Gomori's substrate was used at pH 9.4 but the Gomori procedure was simplified and modified by avoiding the use of alcohols in subsequent procedures. Sections were deparaffinized following incubation in sodium glycerophosphate and subsequent treatment with 2 per cent cobalt chloride solution and ammonium sulphide water. Incubation time was limited to ten to twenty minutes. In the case of the controls, sodium glycerophosphate was omitted from the incubating solution. Sections were last deparaffinized in xylol and mounted with balsam.

OBSERVATIONS

The areas specifically studied were the mandible, Meckel's cartilage, the anterior process of the malleus, and the tympanic ring.

The mandible in the rat is the first skeletal structure to ossify. On the fifteenth day of intrauterine life or slightly earlier a condensation of osteogenetic cells appears on the anterolateral surface of Meckel's cartilage. These cells constitute the primordium and the center of intramembranous ossification of each half of the developing mandible (Fig. 1, *M* and *R*). At this stage these osteogenetic precursors retain the cytological characteristics of their mesenchymal precursors. The centrally located cells of this condensed cellular mass appear modified when compared with the surrounding mesenchyme. The symmetrical Meckel's cartilages, right and left, represent the cartilaginous core of the first visceral arch. They are among the earliest skeletal structures to chondrify and are easily identified at this stage by their young cartilaginous texture. They have undergone no osseous change and no alkaline phosphatase activity is demonstrable. An adjacent section through the mandible stained for alkaline phosphatase (Fig. 2) demonstrates the presence of this enzyme by a black-staining precipitate of cobalt sulphide in the nuclei of these osteogenetic precursors and in a few of the formed osteoid intercellular fibers in the ossification site of the mandible. In contrast, the surrounding mesenchyme remains unstained.

With further development of the bony mandible osteoblastic precursors become more identifiable from the surrounding mesenchyme. This is represented at a sixteen-day stage (Fig. 3, *R*). The bone-forming cells in this region of the upper ramus have become enlarged and cuboid, aligning themselves in osteogenetic rays. This is preparatory to intramembranous-bone deposition at this site of ossification.

Other anlagen of sites of intramembranous ossification are seen at this stage in the tympanic ring and in the anterior process of the malleus (Fig. 3, *T* and *A*). Ossification of the anterior process of the malleus begins in the perichondrium of

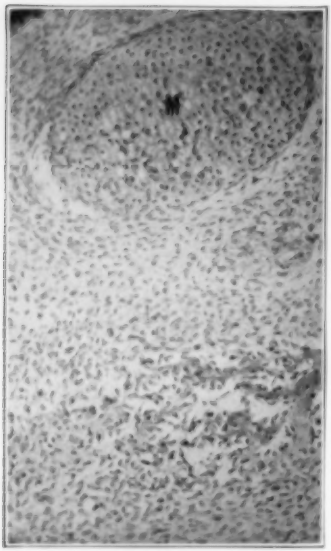

FIG. 5

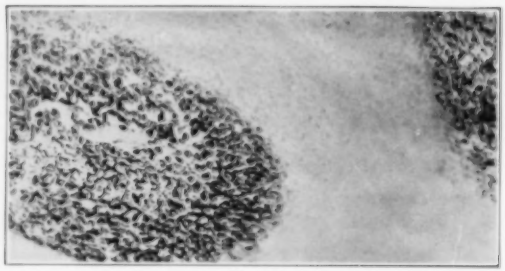

FIG. 6

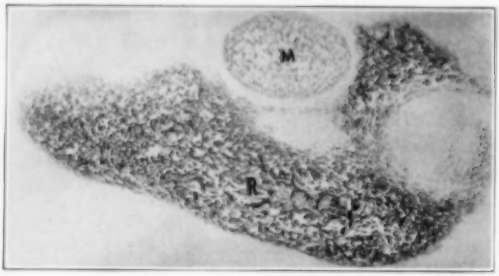

FIG. 7

Fig. 5: Photomicrograph of sixteen-day stage of the body of the mandible stained with hematoxylin and eosin (× 107). Note the non-osseous portion of Meckel's cartilage at *M* and membranous bone trabeculae.

Fig. 6: A higher magnification of Fig. 4 (× 107) showing enzymatic activity in the nuclei and cytoplasm of osteoblasts. Enzyme activity is also present in the preosseous collagen fibers. Note clear demarcation from the general surrounding mesenchymal cells.

Fig. 7: Photomicrograph of sixteen-day fetal mandible (× 54) cut through a region adjacent to the section represented in Fig. 5. Note Meckel's cartilage (*M*) free of enzymatic activity, the developing tooth socket appearing as an island of mesenchymal cells free of enzymatic activity, the ossifying lower ramus of the mandible at *R* with differentiated mesenchymal osteogenetic precursor cells well demarcated from the general mesenchyme.

Meckel's cartilage. In both the tympanic ring and in the anterior process of the malleus this mode of osteogenesis continues into adult form. Meckel's cartilage (Fig. 3, *M*), represented here in its mid-portion, shows no evidence of ossification. An adjacent section stained for alkaline phosphatase (Figs. 4 and 6) clearly demarcates these ossification centers from the unstained and undifferentiated mesenchyme. The activity of the enzyme appears to be localized in the nuclei and in the cytoplasm of osteoblastic precursors in the centrally located cells while nuclear activity alone is demonstrated in the cells along the margins of the well demarcated ossification sites in the upper ramus and tympanic ring. At the periphery of Meckel's cartilage (compare Figs. 3 and 4) black-staining scattered granules appear in the anlage of the ossifying anterior process of the malleus but Meckel's cartilage remains free of osseous changes and alkaline phosphatase activity. Osteoid intercellular matrix surrounded by osteoblasts does demonstrate intense activity. Figures 5 and 7 represent a section from a lower level of the same stage and demonstrate the ossifying body with the same findings as those described at a higher level of the ramus (Figs. 3 and 4). With further development of the bony mandible as in the seventeen-day stage, the upper ramus acquires a preskeletal type of tissue (compare Figs. 3 and 8). It is characteristic of this tissue either to transform directly into bone or to develop into cartilage and then undergo endochondral ossification. The cells of this tissue show gradations between chondroid cells, osteoblasts, and proliferating fibroblasts, some of which have differentiated into an intermediate stage between chondroblasts and

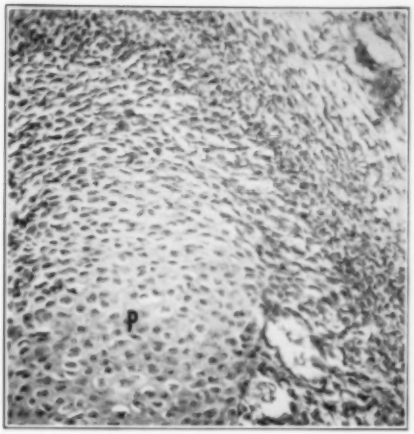

FIG. 8

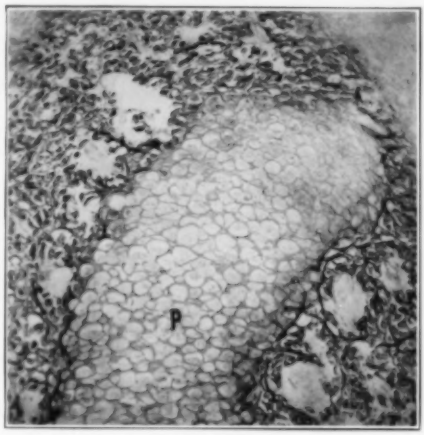

FIG. 9

Fig. 8: Photomicrograph of a seventeen-day fetal rat mandible (× 107) stained with hematoxylin and eosin. Note the preskeletal type of tissue in the developing upper ramus (*P*). Posteriorly, the cells are differentiating to rapidly proliferating osteoblastic cells and anteriorly to chondroid cells.

Fig. 9: An adjacent section (× 107) demonstrating alkaline phosphatase activity in the surrounding osteogenic cells and in the intercellular osteoid matrix. The preskeletal type of tissue (*P*) demonstrates slight cellular and intercellular activity. Its cells at the periphery grade into the surrounding osteoblast and demonstrate relatively more activity than the centrally located cells.

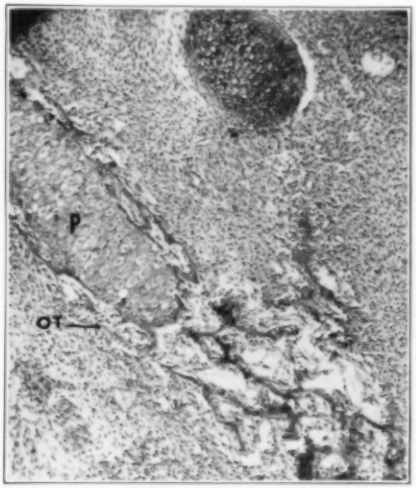

FIG. 10

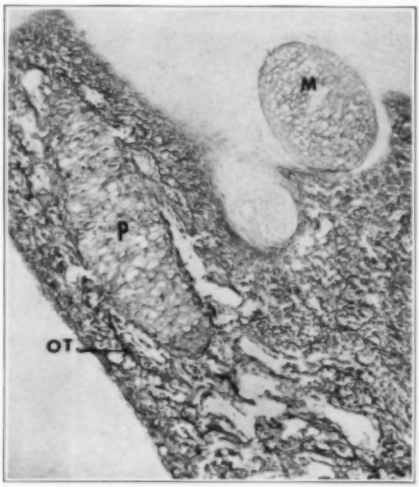

FIG. 11

Fig. 10: Photomicrograph of an older stage than previously, cut through the base of the upper ramus and non-ossifying portion of Meckel's cartilage of a fetal rat mandible, at nineteen days, stained with hematoxylin and eosin (× 107). This slide shows an appreciable amount of membrane bone deposited around the island of hypertrophic cartilage at *P*. Note osteoid tissue (*OT*) developing in preparation for membrane-bone deposition.

Fig. 11: An adjacent section (× 54) demonstrating alkaline phosphatase activity in the cell nuclei and cytoplasm of the hypertrophic cartilage. Around the periphery of this cartilage are bone trabeculae, with osteoid surrounded by osteoblastic cells. Note the intense activity in the osteoid trabeculae and at the margins of the growing bone trabeculae. Meckel's cartilage is free of enzymatic activity, the general mesenchyme is unstained. Osteoid trabeculae (*OT*), Meckel's cartilage (*M*). Preskeletal tissue with hypertrophic cartilage cells at *P*.

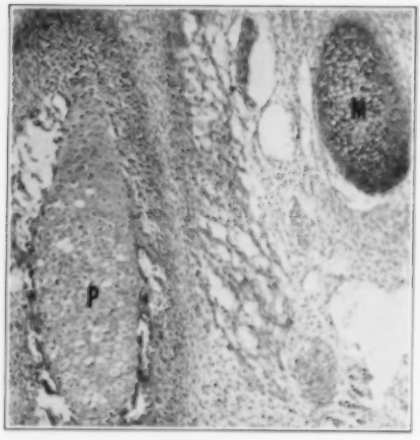

FIG. 12

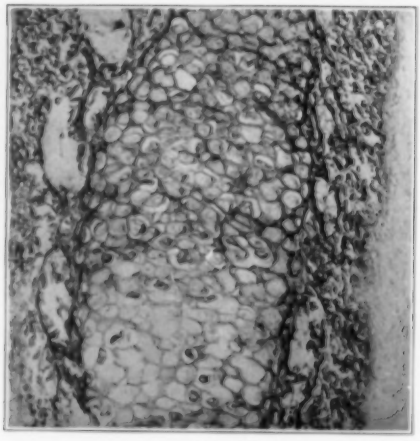

FIG. 13

Fig. 12: Photomicrograph of an eighteen-day rat through the ramus, stained with hematoxylin and eosin (× 56) showing the preskeletal tissue (*P*) surrounded by membranous-bone spicules. This tissue differentiates to osteoblastic precursors on one side and to hypertrophic cartilage in another direction. Note the surrounding rapidly proliferating osteogenetic cells. Meckel's cartilage (*M*).

Fig. 13: An adjacent section stained for alkaline phosphatase activity (× 111). Note the clearly demarcated osteogenetic tissue from the general mesenchymal cells. Also note the enzymatic activity in the nuclei, cytoplasm, and matrix of hypertrophic cartilage cells.

FIG. 14

FIG. 15

Fig. 14: Photomicrograph of a rat fetal mandible, nineteen days, cut at the anterior portion of the lower ramus, showing the ossifying part of Meckel's cartilage (*M*) surrounded by osteogenetic tissue with considerable amount of membrane bone already deposited. Also note the developing site of a tooth. Stained with hematoxylin and eosin (× 56).

Fig. 15: An adjacent section showing alkaline phosphatase activity concurrent with the preosseous changes in Meckel's cartilage. Note the black precipitate in nuclei, cytoplasm, and intercellular matrix. Also note the diminishing activity of alkaline phosphatase in the bone trabeculae as compared with that of osteoid intercellular fibers (× 56).

osteoblasts. Surrounding this tissue is an area of rapidly proliferating osteogenetic cells.

An adjacent section stained for alkaline phosphatase demonstrates nuclear and cytoplasmic enzyme activity both in these bone-forming cells and in the intercellular osteoid matrix (Fig. 9). The newly formed, "neomorphic", preskeletal tissue also shows some activity in the nuclei and in the matrix. At later stages of eighteen and nineteen days (Figs. 10, 11, 12, and 13 at *P*) these cells become typical hypertrophic cartilage cells with associated minimal calcification in the matrix. Alkaline phosphatase studies show nuclear and cytoplasmic, as well as intercellular enzymatic activity associated with the preosseous changes. In these later stages a considerable amount of membrane bone is deposited (Figs. 10 and 12). Adjacent sections stained for alkaline phosphatase (Figs. 11 and 13) demonstrate less activity in the osteoblasts surrounding the bone trabeculae than in the osteoblasts surrounding an osteoid zone. The osteoid laminae on the margins of the bone trabeculae are heavily and intensely stained as compared with the already ossified trabeculae.

The association of alkaline phosphatase in ossifying cartilage of endochondral type is well demonstrated in a nineteen-day stage (Figs. 14 and 15). The cartilaginous structure enveloped by the developing membranous bone of the lower ramus of the mandible is Meckel's cartilage. At this stage Meckel's cartilage undergoes osseous alteration along its anterior portion as demonstrated in Figure 14 at *M*. An adjacent section shows enzymatic activity in the nuclei and cytoplasm of the hypertrophic cartilage cells and in calcifying matrix. This resembles the hypertrophic cartilage present in the developing upper rami (compare Figs. 12, 13 at *P*, 14, and 15 at *M*). The remainder of Meckel's cartilage does not show enzymatic activity (compare Figs. 1 through 7, 10, 11, and 12).

DISCUSSION

The physiochemical mechanisms involved in chondrification and ossification are not as yet completely understood. Of the various factors involved in these biological phenomena one is alkaline phosphatase.

The early signs of enzymatic activity in membrane bone appear concurrently with condensation of osteogenetic mesenchymal cells (Figs. 1 and 2). The enzyme first appears in the nuclei and then with further differentiation of these osteogenetic precursor cells the enzyme appears in their nucleoli and their cytoplasm (Figs. 4, 6, and 7). With further development of osteoblasts and the appearance of osteoclasts the enzyme becomes extracellular. Mineral salts are then deposited among the preosseous collagen fibers or osteoid matrix, and the phosphatase activity becomes weaker or disappears except in the osteoid laminae at the margins of the growing bone trabeculae (Figs. 12, 13, 14, and 15).

In contrast to this, alkaline phosphatase is not associated with the early condensation of mesenchymal cells at the start of chondrogenesis. It does not become demonstrable until hypertrophic and calcific changes are present. At the onset of enlargement or hypertrophy of cartilage cells, phosphatase activity is noted in the nuclei and then it gradually appears first in the cytoplasm and then in extracellular matrix, the amount depending upon the degree of hypertrophy and the extent of the deposition of inorganic salts in the altered cartilaginous matrix (Figs. 14 and 15). The constant association of this enzyme with the osseous change suggests that it plays some specific role in bone formation. Meckel's cartilage, as previously described, ossifies in its anterior portion and the remainder dedifferentiates into connective tissue. In this study it has been noted that the non-ossifying part of Meckel's cartilage is free of enzymatic activity in contrast to its anterior ossifying portion (compare Figs. 4, 5, 7, 11, and 15).

The preskeletal type of tissue seen in the ramus of the mandible appears from the standpoint of histogenesis to be an alternate product of osteogenetic stem cells (Figs. 8 and 9) which may go directly to bone or give rise to a type of

tissue which resembles hypertrophic cartilage. This tissue undergoes calcific and hypertrophic changes but it does not originate in a manner similar to other chondrocytic structures which normally go through a progressive differentiation of mesenchymal cells toward typical chondrocytes. This is evident if one compares Figures 3 and 8. In its early histogenesis this intermediate type of skeletal tissue demonstrates slight enzymatic activity. Later as osteoblastic and preosteoblastic cells appear intense nucleolar and cytoplasmic alkaline phosphatase activity are found. With further development, this tissue (Figs. 10 and 12) shows characteristic osseous changes resembling those in a cartilaginous structure undergoing changes preparatory to endochondral ossification. Alkaline phosphatase studies at this stage reveal enzymatic activity in the nuclei, cytoplasm, and intercellular matrix (Figs. 11 and 13).

As to the role of alkaline phosphatase activity in bone formation, it may be stated in view of our findings that this enzyme plays a local and specific part in the osteogenesis of bone, calcification of cartilage, and differentiation of mesenchymal tissue to osteogenetic or osteoblastic cells.

SUMMARY

1. Meckel's cartilages and the intramembranous sites of ossification in the mandible, tympanic ring, and the anterior process of the malleus of fetal rats between fifteen and twenty days of intrauterine life have been studied with respect to the localization of alkaline phosphatase during the normal differentiation of embryonic mesenchymal cells to osteoblasts and chondroblasts.

2. Alkaline phosphatase appears in the nuclei concurrent with the preliminary condensation of osteogenic mesenchymal cells in the primordium of an intramembranous ossification center.

3. With further differentiation of these osteogenetic precursors to bone-forming cells, the enzyme appears in the nucleoli and in the cytoplasm. At an osteoblastic stage it also appears intercellularly in the osteoid matrix.

4. Mesenchymal cells that have not differentiated in an osteogenetic direction are free of enzymatic activity in contrast to the mesenchymal osteogenic precursor cells.

5. No enzymatic activity was noted during the condensation and differentiation of mesenchymal cells in a chondrocytic direction until hypertrophy of chondrocytes took place preparatory to ossification.

6. Enzymatic activity was demonstrable in the ossifying anterior portion of Meckel's cartilage but not in its non-ossifying portion.

7. The preskeletal type of tissue, an intermediate between bone and cartilage, which may also be called secondary or hypertrophic cartilage, occurs in the ramus of the membranous bony mandible. This is a "neomorphic" product in the differentiation of rapidly proliferating osteogenetic or osteoblastic cells in a site of intramembranous ossification. This is well demonstrated by our studies of alkaline phosphatase activity.

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DISCUSSION

Treatment of Giant-Cell Tumor of Bone

(*Continued from page 904*)

argument, although the authors provide good evidence to show that it does. That it is not an effective method of treatment, however, seems to be demonstrated. It failed in all thirteen cases in which it was used as a primary method of treatment. It was used at some time in seventy-four of the 116 cases in combination with other treatment. The poor results in most cases and the complications resulting in others should discourage its use in all but those lesions situated in the flat bones and cancellous bones where no other type of therapy is possible.

I am willing to advise curettage, after chemical cauterization and packing with bone chips, as a primary method of treatment for a giant-cell tumor presenting in a long bone which enters into the formation of a major articulation. The authors' report indicates that success with curettage will be obtained in less than half of the cases, maybe about 40 per cent.

If the tumor recurs, then I strongly favor complete resection and reconstruction of the extremity by bone graft, arthrodesis, and intermedullary-nail fixation. For all giant-cell tumors involving small bones I favor resection as a primary method of treatment where it can be carried out with little functional loss. If we are to improve our results in the treatment of giant-cell tumors, orthopaedic surgeons must develop a more aggressive attitude toward treatment.

We have had one recurrence with metastasis in a giant-cell tumor that had been given three previous curettage treatments and then was followed by a resection. This tumor was at the lower end of the radius, and within a year there was recurrence in a small fragment of the distal end of the ulna that had been left in and also in the stump of the radius which still remained. In this case we did an amputation above the elbow, and, later, pulmonary metastasis developed. I am quite sure that in this case the recurrence was due to transplantation of tumor tissue at the time of the resection; the shell must have been broken into and tumor tissue transplanted on the instruments and remained. I think that this contingency must be borne in mind. This case certainly proved to my own satisfaction that we must be very careful to stay outside tumor tissue and, if we do break in, we should take steps to change our instruments, to clean the area, and do all that we can to prevent the possibility of transplantation of tumor tissue.

Osteoid Osteoma of a Cervical Vertebra

Report of a Case

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Osteoid osteoma is a small, benign bone lesion, affecting a single bone and characterized by a specific clinical and roentgenographic pattern. The disease was named and established as an entity in 1935 by Jaffe [3]. The primary component or nidus is an oval structure, usually not more than one centimeter in diameter, which is composed of osteoid and atypical bone. The nidus is, classically, surrounded by a zone of dense reactive bone of varying width.

Dull, aching pain, worse at night and frequently relieved by aspirin, and point tenderness over the lesion are important diagnostic features. Roentgenographic examination revealed the nidus as a radiolucent, oval area surrounded by sclerosis. A variable degree of nidus calcification is occasionally noted [4,5].

Osteoid osteoma has been found in almost every bone [7] except the skull. The number of reported cases exceeds 200. Recently, Sabanas and associates studied the natural history of this lesion in the spine. Its presence in this location is not rare; the literature contains reports of thirty-six cases. According to the authors, the laminae of the vertebrae are most often involved, although any portion of the posterior segment can be affected [5,9].

However, osteoid osteoma rarely appears in a vertebral body primarily. Walker reported the case of a fourteen-year-old boy in whom a nidus was discovered in the body of the fifth cervical vertebra. The pedicle was secondarily involved in that case.

Benign osteoblastoma (giant osteoid osteoma) has a predilection for the spine, and has been observed in the vertebral body [1]. This lesion is distinguishable from osteoid osteoma by its larger size, roentgenographic appearance, and destructive nature.

The purpose of this report is to present a case of osteoid osteoma in an unusual site—the body of a cervical vertebra.

E. D., a white boy, nine and a half years old, was admitted to St. Charles Hospital on November 20, 1957, because of pain on the left side of his neck, tenderness, and torticollis of four years' duration. The pain was confined to the left upper cervical region and did not radiate. It was intermittent and dull, tending to become more severe in the late afternoon or evening. Relief could not be obtained by rest but salicylates were effective. Progressive spasm of the muscles of the neck with accompanying torticollis, generally poor posture, sagging of the shoulders, and dorsal round-back deformity were noticed by the parents. The past history was not contributory.

The positive physical findings were as follows: The head was inclined to the left approximately 20 degrees, and the active range of motion in all spheres was 50 per cent of normal. The left sternocleidomastoid muscle was tense but there was neither apparent scoliosis nor palpable masses. Exquisite tenderness was present at the angle of the mandible in a well defined area about one inch in diameter. There was no local swelling, heat, or change in skin color; the cervical nodes were not enlarged.

Although roentgenograms were made in several projections, the oblique tomogram proved to be of most value (Fig. 1). This demonstrated an area of mottled density, measuring one by one centimeter, in the inferior lateral portion of the third cervical body. There was a surrounding radiolucent zone and marked sclerosis of the adjacent bone.

Laboratory studies included complete blood count and determination of sedimentation rate, blood proteins, calcium, phosphorus, and alkaline phosphatase. All were within normal limits.

Operation was performed seven days after admission. A transverse skin incision was

FIG. 1

FIG. 2

Fig. 1: Oblique tomogram of the cervical spine showing sclerosis of the vertebral body and the nidus surrounded by a radiolucent halo.

Fig. 2: Oblique roentgenogram made by routine technique thirteen months postoperatively. Note bone regeneration.

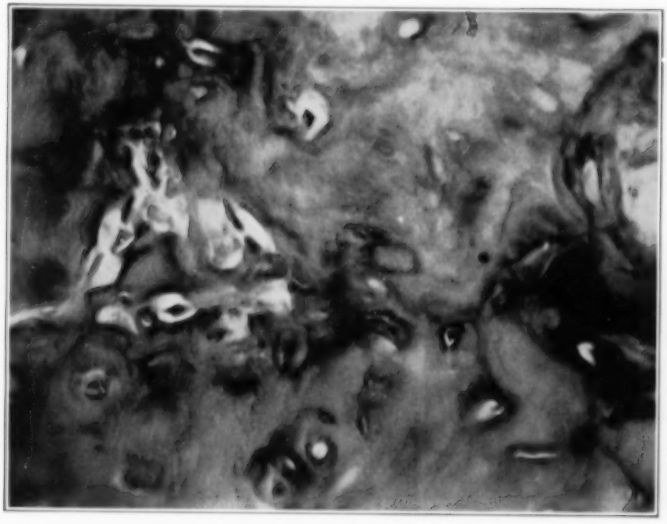

FIG. 3

Photomicrograph (× 400). A representative field taken from the operative specimen, demonstrating osteoid, irregular bone formation, and variable calcification.

made in the anterior carotid triangle overlying the third cervical vertebra and the deep cervical fascia was opened longitudinally. Dissection was then directed to the interval between the neck viscera, medially, and the carotid sheath and sternocleidomastoid muscle, laterally. An endotracheal tube which had been placed in the esophagus was helpful in demonstrating the exact extent of the lateral wall of that structure. The periosteum was then reflected from the vertebral body; the uncovered bone appeared sclerotic. Approximately

one square centimeter of bone was removed after roentgenograms confirmed the location of the lesion. During curettage of the superficial bone, a small, yellow, granular and discrete body which proved to be the nidus was easily detached and taken as a specimen. Esophageal drainage and examination of the wall indicated that the viscus had not been inadvertently perforated and the operation was concluded by closure of the skin.

The specimen removed consisted of a waxy, gray fragment, measuring 0.5 by 0.5 centimeter, which microscopically demonstrated compact, irregular islands of poorly formed partially calcified osteoid tissue together with scattered multinucleated giant cells. The diagnosis was nidus of osteoid osteoma (Fig. 3).

The postoperative course was uncomplicated. The sutures were removed on the seventh day, and gentle neck stretching was begun by a physical therapist. The patient was discharged on the twenty-first day.

When last seen in December 1958, thirteen months postoperatively, the child was completely asymptomatic. Neck motion was still limited to 75 per cent of normal because of contracture of the sternocleidomastoid. A follow-up roentgenogram (Fig. 2) showed no recurrence.

DISCUSSION

This patient had the second reported case of osteoid osteoma in the body of a vertebra. The pain suggested osteoid osteoma but the physical examination was not specific except for localized "finger point" [2] tenderness. Torticollis probably developed in response to pain initially as the patient attempted to splint the left side. Contracture of the sternocleidomastoid muscle, due to the prolonged illness, is not surprising. The most important diagnostic clue was provided by the roentgenograms. Oblique and tomographic views of the neck revealed the lesion with clarity. Because of its central position, a direct approach to the lesion was necessary [8]. The nidus was accurately outlined and readily removed through an anterior approach.

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Metaphyseal Chondrodysplasia of Jansen

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In 1934 Murk Jansen presented a most unusual case of disturbed endochondral bone formation under the name *metaphysäre dysostosis*. His patient was a German boy born with bilateral club-foot in whom swelling in the joints gradually developed. He began to walk after the age of three and then only with the help of braces. At ten years of age he was twelve inches below normal height. Roentgenograms showed that the metaphyses were enlarged and were still cartilaginous. There were numerous spots of calcification and ossification scattered throughout the metaphyses. The diaphyses were thickened and some were curved; their ends were irregular and expanded. When the boy was five years old, his serum calcium was 16.6 milligrams per 100 cubic centimeters and when he was ten, it had fallen to thirteen milligrams per 100 cubic centimeters. Determinations of other constituents of the blood and urine were said to be within normal limits, but what these tests were is not reported.

Jansen's term *metaphysäre dysostosis* (or metaphyseal dysostosis) has little to recommend it as a name for the disease, since it states in only the broadest term that a defect in bone formation is present. The abnormality is a persistence of epiphyseal cartilage and a defect in the formation of endochondral bone. Metaphyseal chondrodysplasia of Jansen is therefore proposed as a more appropriate title. Although there are objections to the eponymy, in this rare disease it has the advantage of specifying one of a group of similar abnormalities.

Jansen considered the cause of this disease to be abnormal intra-uterine pressure acting at a particular stage of fetal development. Fairbank classified the disease under congenital developmental errors; in his opinion, Jansen's was the only case reported in the literature up to 1951. Fairbank mentioned having seen two cases of widespread metaphyseal dysplasia similar, but not exactly comparable, to Jansen's case. Cameron, Young, and Sissons, in 1954, reported what we believe to be an example of a metaphyseal chondrodysplasia in a seven-year-old Chinese boy. In this patient the serum calcium was 10.0 to 13.7 milligrams per 100 cubic centimeters; the serum inorganic phosphorus, 3.4 to 4.9 milligrams per 100 cubic centimeters; and the alkaline phosphatase, 16 to 20 King-Armstrong units per 100 milliliters. Another case was described by Grabe of Heidelberg in 1931, but the diagnosis was not made until recently, when it was reported by Hohmann and associates, in 1957. Laboratory data were not given. Follow-up study showed that after twenty-six years the patient had remained a dwarf, with limited motion of all joints and, significantly, that the uncalcified masses of cartilage had all become ossified although the metaphyses maintained their enlarged contours. Other reports of cases, which were termed metaphyseal dysostosis [3,13,15,18] have appeared but, in our opinion, differ fundamentally from the condition under consideration.

To the best of our knowledge, the patient reported here is the fourth case to be described, and the first in the American literature. It is felt that many of these cases have been considered to be forms of Ollier's disease and other anomalies. Our case resembles those of Jansen, Cameron and associates, Grabe [10], and, in addition, presents interesting biochemical abnormalities.

L. D. M. was born, at term, weighing six pounds and fifteen ounces, after an uncomplicated pregnancy and delivery. The parents and three siblings were normal, and there was no family history of dwarfism or other skeletal abnormality. When the infant was two months

FIG. 1-A

FIG. 1-B

Figs. 1-A and 1-B: Roentgenograms made when the patient was seven weeks old.
Fig. 1-A: Note the early appearance of dysplasia at the wrist and elbows.
Fig. 1-B: Roentgenogram showing the symmetrical changes at the metaphyses. Similar, although not identical, changes are sometimes seen in infantile hyperparathyroidism.

FIG. 2-A

FIG. 2-B

FIG. 2-C

Figs. 2-A, 2-B, and 2-C: Patient at five years of age.
Fig. 2-A: The dwarfism involves the lower extremities more severely than the upper extremities.
Fig. 2-B: Side view showing the standing posture. The child measured only 11.6 inches from pubis to heel.
Fig. 2-C: The characteristic swelling of the metaphyses produced a depression at the joint lines.

FIG. 3

FIG. 4

Fig. 3: Roentgenograms of the left lower extremity. The chondrodysplasia involves only the metaphyses, the epiphyses being relatively normal. (The right lower extremity showed identical changes.)

Fig. 4: Roentgenogram showing symmetrical metaphyseal involvement of the hips. The pelvis is severely involved.

FIG. 5

Fig. 5: Roentgenograms of the hands show that, characteristically, the chondrodysplasia is adjacent to the epiphyses and confined to the metaphyseal areas. There is retardation of ossification of the carpal bones.

of age she was admitted to another hospital because of vomiting and loss of weight. It was evident that a generalized skeletal defect was present, and pathological fracture of the femur was suspected because of deformity. A skeletal survey revealed angulation of the long bones; decreased density of the metaphyses; cupping of the distal ends of the bones, which were of ragged appearance; and generalized osteoporosis, including the skull (Figs. 1-A and 1-B). The

epiphyseal cartilages were thickened and the ribs showed marked angulation and deformity. There was no evidence of pathological fracture. The diagnoses considered at this time were Morquio's disease, combined rickets and scurvy, osteogenesis imperfecta, and renal rickets. No definite diagnosis was established. The available laboratory data did not include estimations of serum calcium, phosphorus, or alkaline phosphatase. On two occasions the Sulkowitch test showed a moderate amount of calcium present in the urine.

The infant remained in the hospital for three months, during which time she was critically ill with repeated attacks of tracheobronchitis and otitis media. At five months of age she had regained her birth weight and was discharged from the hospital. She received supplementary vitamins during hospitalization and after discharge. After the infant was six months

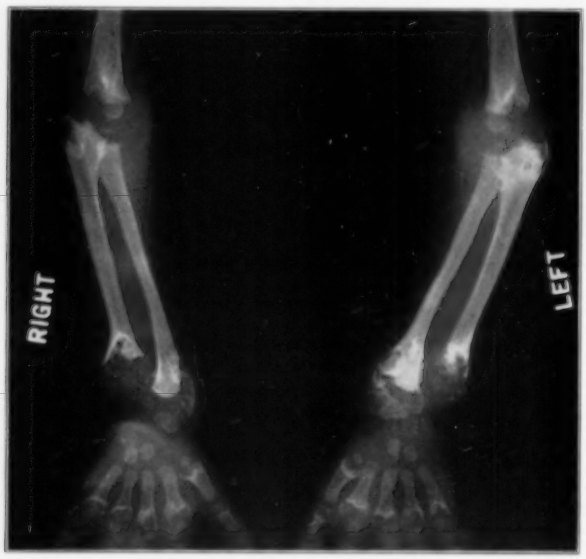

FIG. 6

Roentgenogram of the elbows shows only mild involvement.

FIG. 7

Fig. 7: The pattern of metaphyseal chondrodysplasia in the feet is identical with that seen in the hands. The dysostosis is proximal in the first metatarsal and distal in the others. The tarsals are not involved, except for the calcaneal epiphysis.

of age her general health improved and she gained weight slowly. At one year she weighed eleven pounds; at two years, fifteen pounds; and at three, eighteen pounds. Her height increased from twenty inches at birth to thirty inches at three years. At four years of age her weight was twenty-two pounds and her standing height, twenty-eight inches (recumbent height, 31.2 inches). She sat unaided at the age of two, but did not walk until she was three. She was talking by the time she was two years of age and was considered by her family to be of normal mentality.

The child was first seen by us when she was five years and four months old, after the family had moved to Detroit. The patient was a dwarf, with short legs and relatively long arms (Figs. 2-A and 2-B). She measured twenty inches from head to pubis, and 11.6 inches from pubis to heels. The circumference of her chest was 16.4 inches and of her abdomen, 19.2 inches. All of these measurements fell below the third percentile for her age. When she stood, her hips and knees were held in marked flexion although there was no actual flexion deformity. The lower extremities appeared to be bowed anteriorly owing to deformities of the metaphyses. Her gait was waddling and laborious; marked rotation of the pelvis and legs accompanied each step. Her patellae were dislocated laterally. All weight-bearing joints were stable and had a good range of motion. There were round, smooth, non-tender symmetrical enlargements at the knee, so that a depression appeared at the joint line (Fig. 2-C). The skull was macrocephalic with hypoplastic facial bones. The fontanels were closed and the chin was receded. The eyes were widely spaced and exophthalmic. The teeth were normal and did not show hypoplasia of the enamel. The cornea showed no calcium or cystine crystals. There were no masses in the neck. The chest was bell shaped, and no enlargement of the costochondral junctions was evident. The cardiorespiratory and neurovascular systems were normal on physical examination. The blood pressure was 100 systolic and 40 diastolic. A psychometric examination revealed an intelligence quotient of 75. There was moderate retardation of motor coordination.

ROENTGENOGRAPHIC ASPECTS

In the long bones the epiphyses, although somewhat enlarged, were relatively normal in appearance. The diaphyses were short and broad with some coarsening of the trabecular pat-

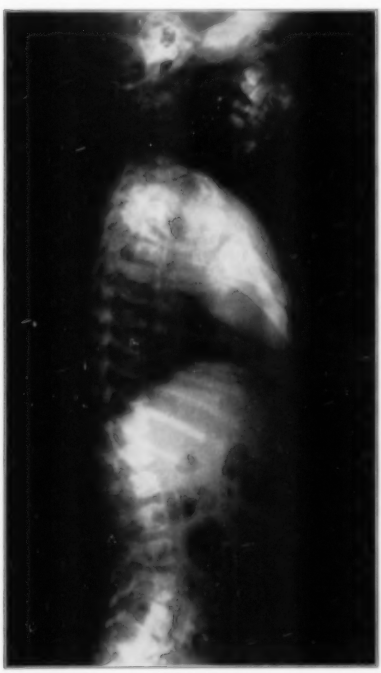

FIG. 8

FIG. 9

Fig. 8: This roentgenogram shows the spine to be normal.

Fig. 9: Dysplasia is present at the vertebral ends of all the ribs. The calcific areas in the region of the twelfth ribs represent nephrocalcinosis. This was proved by an intravenous pyelogram.

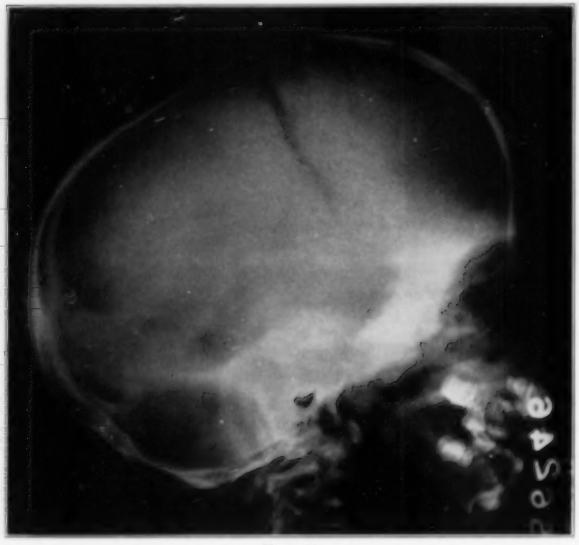

FIG. 10

The skull shows craniostenosis, loss of diploic bone, platybasia, and macrocephaly.

tern. For the most part, however, the diaphyses were not markedly abnormal. Osteoporosis or pseudofractures were not observed.

The metaphyses of the long bones and of the bones of the hands and feet showed the following changes:

1. Large, irregular, clear areas of uncalcified cartilage adjacent to the epiphyseal line and under the thin cortical margins of the expanded metaphyses;
2. Patchy, irregular, denser areas representing calcified cartilage in the metaphyses;
3. Areas of irregular ossification near the diaphysis;
4. Wide and irregular epiphyseal lines with relatively normal epiphyses.

BIOCHEMICAL ASPECTS

Very interesting and unexpected biochemical changes were observed in this patient; persistent hypercalcaemia, hypophosphataemia, a consistently elevated serum alkaline phosphatase, and moderately increased urinary excretion of calcium and phosphorus, according to the criteria of Fanconi. In addition, the renal tubular reabsorption of phosphorus was decreased. These changes in the calcium and phosphorus determinations were identical with those found in hyperparathyroidism.

The pH of the urine was repeatedly acid. Renal excretion, as judged by the intravenous pyelogram, was good. The phenolsulphonphthalein excretion was 59 per cent in two hours. The urea clearance was slightly reduced (45 cubic centimeters per minute). The non-protein nitrogen was normal on three determinations, but on one occasion was somewhat elevated (50 milligrams per 100 cubic centimeters). The serum creatinine was consistently low-normal. The serum carbon dioxide was minimally depressed (21.7, 18.3, 21.1, 22.1—normal, 22-27 milliequivalents per liter). The serum chlorides were high-normal (107 and 110 milliequivalents per liter). A urine culture showed numerous colonies of *Staphylococcus aureus* (coagulase positive) on two occasions. The urine was negative for tyrosine crystals. A twenty-four-hour urine specimen was negative for glucose. Twenty-four-hour alpha amino-acid nitrogen excretion was normal (22.5 milligrams).

The serum vitamin A was normal (39 and 30 micrograms per liter). The serum carotenes were 65.4 and 100 (normal, 100-300 micrograms per 100 cubic centimeters). The plasma ascorbic acid was 0.75 and 0.85 microgram per 100 cubic centimeters (normal, 0.7-1.5). The glucose tolerance curve was flat (127 micrograms per 100 cubic centimeters fasting, 118 in 30 minutes, 80 in 90 minutes).

All other laboratory determinations—Wassermann, serum sodium and potassium, cephalin flocculation, bromsulphalein, and cholesterol—were within normal limits. There was moderate hypochromic anaemia. The twenty-four-hour output of 17-ketosteroids was 1.04 milligrams and of hydroxysteroids, 5.7 milligrams. These were considered normal for a child of this age.

TABLE I

THE DISTURBANCE OF CALCIUM AND PHOSPHORUS METABOLISM, JANUARY 1957 TO APRIL 1958

Serum Calcium *Mg./100 c.c.* (Normal: 9 to 11)	Serum Phosphorus *Mg./100 c.c.* (Normal: 4 to 6)	Alkaline Phosphatase *Bodansky Units* (Normal: 5 to 12)	24-Hour Urine Calcium *Mg./kg./day* (Normal: 6.7)	24-Hour Urine Phosphorus *Mg./kg./day* (Normal: 14)	Phosphorus Tubular Reabsorption Test *Per Cent* (Normal: 85 to 90)
13.8	2.2	28.2	49 4.9 mg./kg.*	453 45.3 mg./kg.*	53
12.2	3.2	24.6	113 11.3 mg./kg.*		
13.2	3.4	7.4			
14.4	3.2	12.2			
13.6	3.2	16.8	109 10.9 mg./kg.*	246 24.6 mg./kg.*	58
12.0	2.8	18.8	80 8.0 mg./kg.	189 18.9 mg./kg.*	

*Fanconi stated that the calcium to phosphorus ratio should be 1:2. Our patient showed ratios of 1:4, 1:2.5, and 1:2.2.

PATHOLOGY

A biopsy of the distal femoral metaphysis and epiphyseal line showed thin fascia overlying a thickened periosteum. The cortex was very thin. A specimen was removed which appeared cartilaginous and soft. The peripheral margin was partially covered by a thin layer of bone and partially by fibrous tissue, poor in cells.

The main portion of the specimen was mature hyaline cartilage with uniform cells lying singly in separate lacunae. The cells had no oriented pattern except at the periphery where there was grouping of cells in parallel alignment. There were scattered small areas of calcification and ossification. The surface of the marginal cartilage was irregular and endochondral ossification was irregular. The over-all appearance resembled that of an epiphyseal cartilage with its adjoining center of ossification (Fig. 11).

COURSE

Our original investigation was carried out in January 1957. Leg braces did not appreciably help the child. One episode of flank pain occurred, but no stone was passed.

In April 1958 the patient was reinvestigated. Essentially the same clinical, roentgenological, and biochemical findings prevailed. A parathyroid exploration was carried out because of the biochemical indications of hyperparathyroidism. Four normal parathyroids were present and there was no adenoma. The mediastinum was not explored.

The hypercalcaemia contra-indicated the use of large doses of vitamin D. Empirically, the child was given oral phosphorus feedings * in an effort to correct the hypophosphataemia. For several months this treatment was followed without objective evidence of improvement.

DISCUSSION

The presence of disturbances in the growth and development of bones, accompanied by abnormalities in the serum content of calcium, phosphorus, and alkaline phosphatase stimulated us to study this patient more thoroughly from the metabolic point of view and to report our findings. In children, only a few diseases are characterized by generalized skeletal abnormalities accompanied by well defined metabolic defects. Hyperparathyroidism [1] presents a roentgenographic picture similar to that recorded in this case when the patient was seven weeks old. The hypercalcaemia, hypophosphataemia, hyperphosphatasaemia, and impaired tubular reabsorption of phosphorus associated with nephrocalcinosis are common to both conditions. However, findings in the skeleton, which are not seen in hyperparathyroidism, later appeared in our patient. The findings are: irregularities in

*Disodium acid phosphate, 98 grams, and monosodium acid phosphate, 28.7 grams to make two liters. The dosage was 10 cubic centimeters three times a day, gradually increasing to the limit of tolerance (diarrhoea).

FIG. 11

Biopsy specimen, × 25 (see text).

the ossification of the epiphyseal cartilage; normal density of the diaphyses in roentgenograms; and marked disturbance of epiphyseal growth. Surgical exploration of the parathyroid glands revealed no detectable abnormality. For these reasons, this case cannot be considered one of hyperparathyroidism.

The presence of renal involvement in this case raises the problem of whether the changes in the skeleton may have been secondary to disease of the kidney. Ordinary, secondary hyperparathyroidism of renal origin is generally accompanied by hypocalcaemia or by normal serum calcium, whereas the serum phosphorus generally is elevated. However, it is not possible, at the time of writing, to determine unequivocally whether the renal or the skeletal lesion, in this case, is primary. It seems probable, however, that the continued hypercalcaemia will further damage the kidneys and may result in renal acidosis and progressive renal failure.

The other diseases in which changes in the serum are combined with generalized skeletal abnormalities also do not fit the case in question. Hypervitaminosis D, sufficient to cause hypercalcaemia, was ruled out by the negative history. Vitamin-D-resistant rickets, more correctly called phosphatic diabetes (Dent's Type I renal tubular rickets) resembles this entity in having a low serum phosphorus and low renal tubular reabsorption of phosphorus, but is ruled out by the elevated serum calcium and the inconsistent pathological and roentgenographic findings. Renal tubular acidosis similarly does not show the skeletal findings reported in this case. The syndrome of Fanconi was ruled out by the absence of glucosuria and amino-aciduria. Numerous abnormalities of the skeleton bear a superficial resemblance roentgenologically to this condition in one way or another [16]. Ollier's disease, chondrodystrophia calcificans congenita, and hereditary metaphyseal dysplasia [2] all show skeletal findings at some variance with those reported here and, in addition, do not manifest any metabolic abnormalities similar to those shown by this patient.

Hypophosphatasia [5,17] may show metaphyseal changes similar to those seen in this patient and may also show hypercalcaemia in addition to the hypophosphatasia. In this entity, however, the pathological finding of osteoid is distinctive, and the persistently decreased alkaline phosphatase should differentiate it absolutely from the entity under discussion. Clinical differences in all of the diseases men-

tioned are profound and need not be discussed at length here.

There is no ready explanation for the biochemical abnormalities noted in the case presented. It seems a reasonable possibility that the hypercalcaemia may have resulted in some unknown way from the active metaphyseal disease. Currarino suggested that the hypercalcaemia of hypophosphatasia might be due to failure of normal calcium deposition. Hypercalcaemia itself could then cause nephrocalcinosis which in turn may have resulted in the renal tubular defect. However, renal defects, other than that of reduced tubular reabsorption of phosphorus leading to hypophosphataemia, were not seen in this case.

The explanation just given would suggest that the renal alteration stemmed originally from the bone disease, certainly an unusual situation. This concept is impossible to prove from the existing data. An alternative theory would involve postulating the presence of an undefined metabolic disorder that gave rise to both metaphyseal and biochemical changes. Careful study of future cases may help to elucidate the pathogenesis of this rare disease.

SUMMARY

1. A case is described for which the name metaphyseal chondrodysplasia of Jansen is suggested.

2. Biochemical changes were found simulating those caused by hyperparathyroidism, but different and distinctive roentgenographic findings were also evident. Parathyroid exploration was negative for hyperplasia or adenoma.

3. It is postulated that the metabolic changes—hypercalcaemia and hypophosphataemia—may have been secondary to the underlying bone defect.

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Spontaneous Regression of Reticulum-Cell Sarcoma of Bone

A Case Report

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LOS ANGELES, CALIFORNIA

From the Orthopaedic Hospital, Los Angeles

Since the classic report by Parker and Jackson in 1939, the clinical [3,5,6,10] roentgenographic [10], and histological [6] features of reticulum-cell sarcoma of bone have been presented in detail. Several series [5,6,7,11] and individual case reports illustrating cures by specific therapy [2] have appeared in the literature within recent years. A review of the world literature collecting 154 cases was published in 1957 by Schobinger von Schowingen.

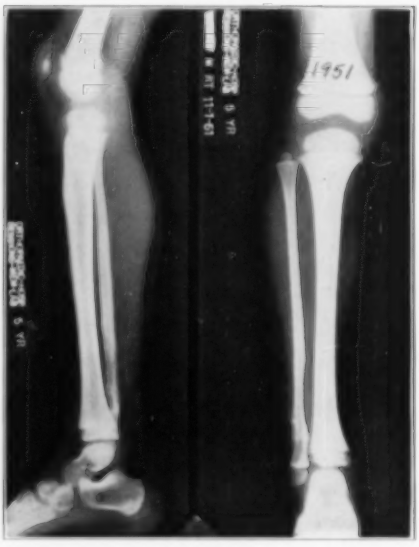

FIG. 1

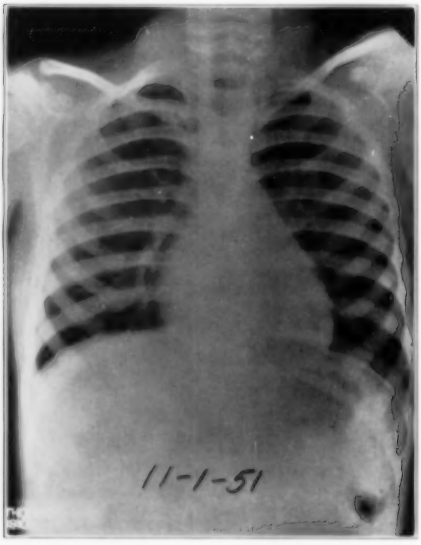

FIG. 2

Fig. 1: Roentgenograms of the entire right tibia and fibula made November 1, 1951, approximately two weeks after the onset of symptoms, revealed a destructive lesion of the distal portion of the fibula characterized by cortical destruction and patchy destruction of the trabecular markings of the medullary canal of the involved bone. Periosteal and subperiosteal reaction and ossification with lamination along the lateral cortex and a small Codman's triangle along the medial cortex were also noted, as well as overlying soft-tissue thickening along the lateral aspect of the fibula.

Fig. 2: Roentgenogram of the chest made November 1, 1951, revealed that the lungs were normal.

The purpose of this report is to present a case of primary reticulum-cell sarcoma of bone, proved by biopsy, which is unique in several respects. The patient, a five-year-old boy, is younger than any found reported in the literature. The youngest patient previously reported was an eight-year-old child in the series of Valls, Muscolo, and Schajowicz. This is also the only reported case of reticulum-

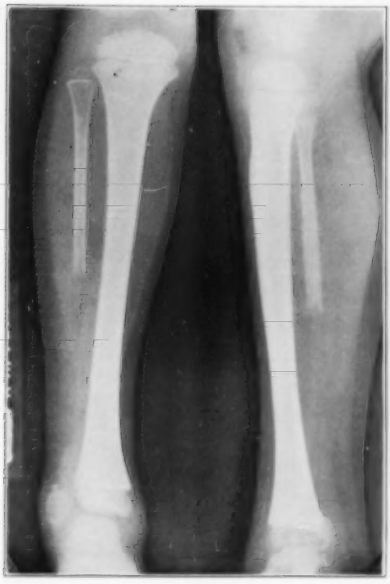

FIG. 3

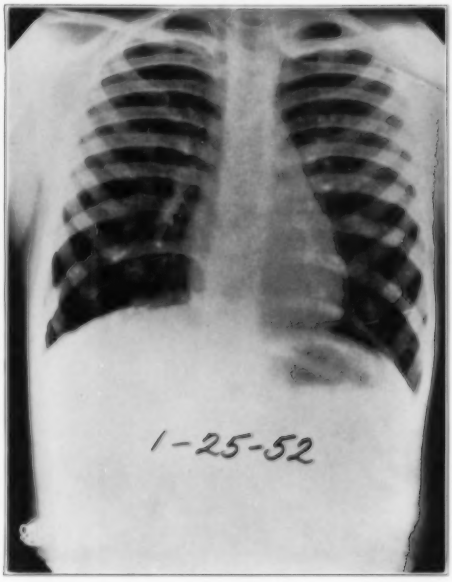

FIG. 4

Fig. 3: Roentgenogram of the entire right tibia and fibula made January 24, 1952, seven weeks after biopsy revealed evidence of surgical resection of the shaft of the distal portion of the fibula, sparing the distal epiphysis. The shaft of the fibula, proximal to the excised portion, showed evidence of internal erosion of the cortex and mottled rarefactions in the medullary canal—findings consistent with tumor involvement.

Fig. 4: Roentgenogram of the chest made January 25, 1952, revealed multiple small areas of infiltration scattered throughout the bases of both lung fields with two or three larger rounded areas of infiltration approximately one centimeter in diameter—findings consistent with a metastatic malignant lesion.

Fig. 5: Roentgenogram of the chest made March 17, 1952, approximately two months after Figure 4, revealed that the previously noted rounded areas of increased density scattered throughout both lung fields had completely disappeared.

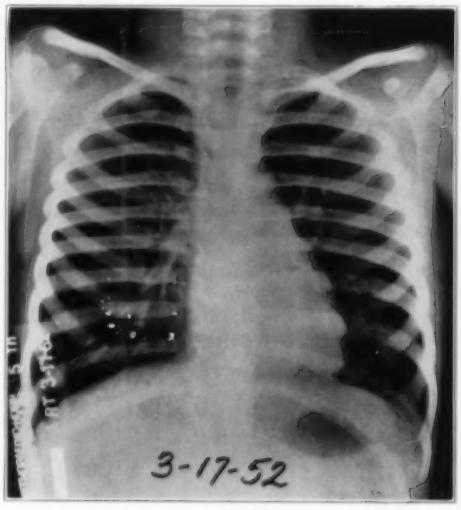

FIG. 5

cell sarcoma, with or without metastasis, which regressed spontaneously. Of reported cases, similar in location and histological appearance, which have regressed, particularly Case 5 of Magnus and Wood's series, Case 7 of Sherman and Snyder's report, and the case of sarcoma reported by Christian and Palmer, all had some form of therapy before regression.

R. J., a five-year-old Caucasian boy, was first seen at the Orthopaedic Hospital on November 1, 1951, complaining of pain and swelling of the outer aspect of the right leg. History revealed that the patient had struck his right leg approximately one month before while playing. He complained of pain only temporarily at that time and there was no noted swelling or discoloration. However, for two weeks prior to admission, the boy had complained of intermittent pain in the right leg; some six days preceding admission the mother had noted

FIG. 6

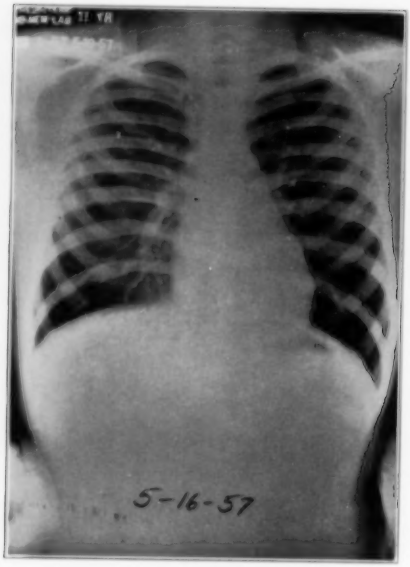

FIG. 7

Fig. 6: Roentgenograms of the right tibia and fibula made May 16, 1957, revealed the absence of the distal half of the right fibula with intact distal fibular metaphysis and epiphysis. The remaining fibula revealed normal bone density without evidence of neoplasm.

Fig. 7: Roentgenogram of the chest made May 16, 1957, revealed no positive findings.

swelling about the painful area without increased heat but with slight pinkness on one occasion. It was further stated that the patient did not complain of pain on weight-bearing, but would not let anyone touch the leg because of pain. The past history revealed no serious illness, injuries, or operations.

Initial examination on admission revealed a well developed, not acutely ill five-year-old boy with normal gait. Positive findings were limited to the right lower extremity where there was a firm swelling about the distal third of the fibula with some overlying skin warmth. There were no palpable lymph nodes in the groin or popliteal fossa on the right. The ankle joint adjacent to the swelling was not swollen or painful to movement.

Roentgenograms of the right leg made the same day (Fig. 1) showed a destructive lesion originating just proximal to the distal epiphysis of the fibula and extending upward for four and one-half inches, with patchy destruction of cortex and medullary canal and a Codman's triangle along the medial cortex. A roentgenogram of the chest (Fig. 2) made at that time revealed the lung fields to be clear.

The white blood count was 12,800 with 54 per cent polymorphonuclear neutrophils, 42 per cent lymphocytes, and 4 per cent eosinophils. The red blood count was 4,390,000 and the hemoglobin, 78 per cent. The sedimentation rate (Cutler) was 18 millimeters in one hour. Serum calcium (one determination) was 14 milligrams per 100 cubic centimeters, serum phosphorus was 4.1 milligrams per 100 cubic centimeters, and the alkaline phosphatase was 10.6 King-Armstrong units.

A working diagnosis was made at that time of a destructive lesion of the distal half of the right fibula consistent with Ewing's endothelioma, osteogenic sarcoma, or osteomyelitis.

On December 6, 1951, a biopsy was done which consisted in resection of the distal half of the diaphysis of the right fibula. At surgery, grayish tumor tissue was found which had infiltrated beyond the bone into the soft tissue about the lower end of the fibula. The cortex was almost completely destroyed, being replaced with necrotic gray material. The distal half of the fibula to the epiphyseal plate was resected with the attached soft tissue. However, because of the malignant appearance of the tumor and the working diagnosis, the procedure was confined to biopsy. No attempt was made to remove all evident tumor tissue, some of which was noted to remain *in situ* at the time of closing the wound. Surgical margin pathological studies were not attempted.

The biopsy report of Dr. Roy Hammack of Los Angeles, now deceased, noted that this was a very cellular tumor, the cells being round or oval with scanty, pale-staining cytoplasm and large, round or oval, vesiculating nuclei. Mitoses were found in moderate number. Sup-

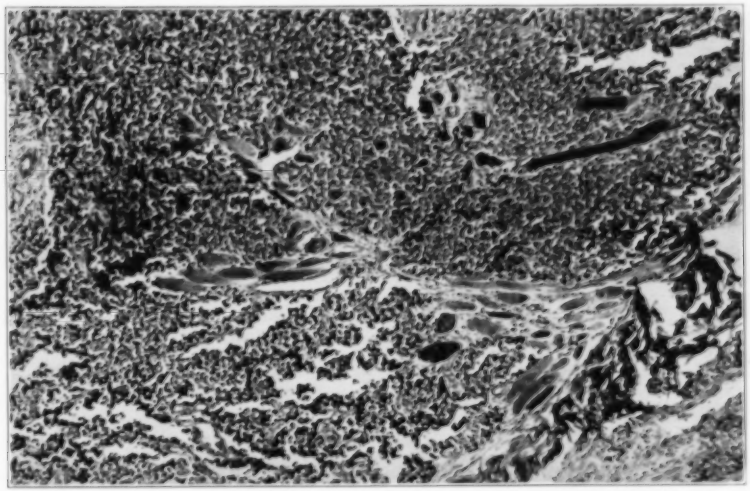

FIG. 8-A

Photomicrograph of the biopsy material showing the marked cellularity of the tumor and the infiltration of the soft tissue by the tumor (hematoxylin and eosin, × 75).

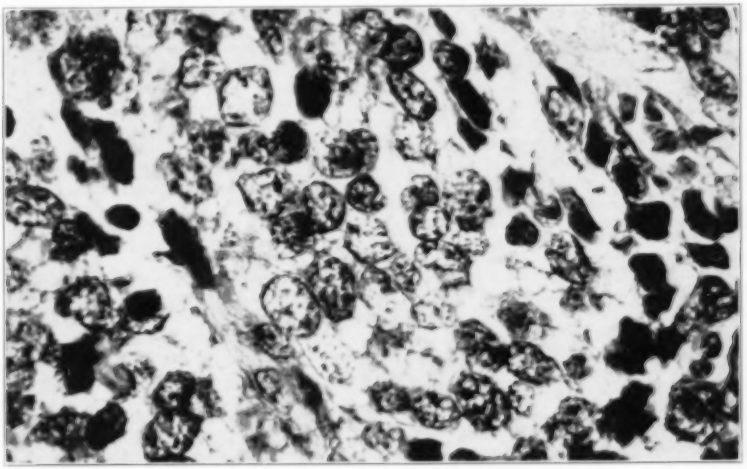

FIG. 8-B

Photomicrograph of the biopsy material showing the characteristic round or oval reticulum cells with scanty, pale-staining cytoplasm and large, round or oval, indented or lobulated, nuclei with prominent nucleoli. The chromatin is coarse. Hyperchromatism is demonstrated in numerous cells (× 1,000).

porting connective-tissue strands contained blood vessels and occasionally the cells formed a radial arrangement about the strands. An occasional blood-vessel wall appeared to be invaded by tumor. In some areas, muscle fibers were enclosed by tumor tissue. Areas of necrosis were frequent. The diagnosis was reticulum-cell sarcoma of the fibula and adjacent tissues.

The postoperative course was uneventful and the patient was discharged from the hospital on December 13, 1951, one week following surgery.

On December 20, 1951, two weeks postoperatively, the sutures were removed from the healed wound and it was recommended that the patient have roentgen therapy directed to

the right distal fibular area. The parents decided to take the boy to the Mayo Clinic for evaluation. On January 9, 1952, a letter was received from the Mayo Clinic with the diagnosis of Grade IV, malignant, small-cell neoplasm, probably atypical Ewing's tumor since the cells were larger than in typical Ewing's tumor. The working diagnosis was changed to Ewing's sarcoma and subsequent recommendations for therapy were based upon that diagnosis.

The patient returned to this hospital on January 24, 1952, asymptomatic except for minimal discomfort about the operative site. No positive clinical findings were noted. Roentgenograms of the right leg (Fig. 3) were made revealing the surgical resection of the distal shaft of the fibula which spared the epiphysis but included the original tumor area. The shaft of the fibula, proximal to the excised portion, showed evidence of internal erosion of cortex and mottled rarefactions in the medullary canal, findings consistent with tumor involvement. At that time, white blood count was 8,800 with 40 per cent polymorphonuclear neutrophils, 50 per cent lymphocytes, 4 per cent transitional cells, 4 per cent eosinophils, and 2 per cent basophils. Red blood count was 4,700,000. Hemoglobin was 80 per cent.

A roentgenogram of the chest was made (Fig. 4) the following day which revealed multiple, small to medium sized rounded areas of infiltration scattered throughout the bases of both lung fields. Because of their bilateral homogeneous appearance, embolic lesions were considered most probable. Since there were no signs of infection, the lesions were thought to be other than septic in origin. Multiple infarcts might have been considered but the roundness of the infiltrates was atypical. The lack of pleural effusion would further militate against such a diagnosis.

Because of the lack of pulmonary symptoms and the diagnosis of Ewing's sarcoma, the roentgenologist interpreted the findings as consistent with metastatic malignant disease.

Palliative therapy was recommended since the diagnosis of Ewing's sarcoma was accepted and it was thought that local as well as metastatic pulmonary spread had already occurred. In order to prevent a large painful tumor during the terminal stages, resection of the remaining right fibula was recommended and roentgen therapy prior to surgery was suggested. The roentgenologist did not feel that roentgen therapy in the presence of pulmonary metastases would alter the clinical situation. Accordingly, roentgen therapy was not given. The local palliative surgery was not carried out and it was decided to continue conservative treatment only.

The patient was next seen on February 14, 1952, two months postoperatively, with a complaint of some local tenderness in the area of the scar. Objective findings were minimal and no therapy was instituted.

On March 17, 1952, three months postoperatively, the boy was seen with an upper respiratory condition of approximately four days' duration. History revealed that it seemed to settle in the chest and cause a non-productive cough. A roentgenogram of the chest (Fig. 5) was made which revealed that the multiple infiltrates found in the previous roentgenogram of the chest had completely disappeared. The patient was then treated conservatively at that time and the cough disappeared in approximately two weeks.

At examination four months postoperatively there was no clinical evidence of local recurrence. Roentgenograms of the leg made then revealed that the distal end of the remaining upper fragment of the fibula had approached the normal.

During the remainder of 1952 the patient's general health remained excellent and there was no clinical evidence of tumor recurrence. Periodic roentgenograms of the right leg made during that year revealed, by ten months postoperatively, that the bone structure of the tibia and remaining fibula was normal. Similarly, frequent roentgenograms of the chest failed to reveal any recurrence of the pulmonary findings. At that time, the case was reviewed and the working diagnosis was changed to reticulum-cell sarcoma with regression of local and metastatic disease.

The patient has been followed at yearly intervals with physical examinations and roentgenograms of the right leg and chest. He was last seen in February 1958, six years postoperatively, at which time a complete skeletal survey was performed in addition to physical examination. His health has remained good and he has remained free of recurrence of the original reticulum-cell sarcoma of the distal portion of the right fibula, clinically and roentgenographically (Figs. 6 and 7).

Photomicrographs of the biopsy material (Figs. 8-A and 8-B) demonstrate the characteristic cellularity, alveolar arrangement of the cells, and the round or oval reticulum cells with scanty, pale-staining cytoplasm and large, round or oval, indented or lobulated, nuclei with prominent nucleoli and coarse chromatin.

In 1957, more than five years after the biopsy was reported and because of the benign course, the original slide and new slides made from the biopsy were reviewed by Dr. J. Vernon Luck and Dr. Weldon K. Bullock of the Orthopaedic Hospital, Los Angeles, and by Dr. Fred W. Stewart of the New York Memorial Hospital. All confirmed the diagnosis of reticulum-cell sarcoma of bone.

This is the youngest patient reported with primary reticulum-cell sarcoma of bone, proved by biopsy. Coin-shaped pulmonary lesions, compatible with met-

astatic malignant disease, were seen roentgenographically; spontaneous regression of the fibular neoplasm and disappearance of the changes in the chest followed. The patient is alive and free of evidence of disease six years following surgery.

We do not claim that pulmonary metastases existed but rather that there was a roentgenographic picture compatible with metastases. Admittedly, no biopsy was performed of the pulmonary lesions. Furthermore, we realize that recurrence with rapid spread of metastases has been reported after five to ten years of apparent freedom from disease. In view of this possibility yearly examinations are being made.

By reporting this case, we are not implying that the therapy described in the previously mentioned similar cases [2,7,10] was not effective; we feel that spontaneous regression of this neoplasm does occur, perhaps due to some natural body defense, not yet demonstrable [4], against tumor growth.

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Orthopaedic Research and Education Foundation

Editorial

Last year a nationally syndicated newspaper columnist devoted an entire column to a critical attack on the numerous public appeals for funds to support research and educational work on behalf of almost every disease and ailment known to man. The author stated that the public was getting sick and tired of so many solicitations of this kind and broadly hinted that some of these were rackets. Perhaps many doctors themselves, appalled by the stream of appeals that flow through the mails, the press, radio, and television channels, may share these sentiments.

One statement, however, was particularly disturbing. The author severely criticized the doctors for not giving more financial aid to the research and development work of their own profession. He implied that the medical profession was relying far too much on the public to finance work which the profession itself should be supporting.

As members of the profession our first inclination might be to rise in indignation to deny this accusation, but on more mature reflection it might be best for us first to look at our own branch of medicine—orthopaedics—to see if our house is in order. Can we "point with pride" to what has been done or should we "view with alarm" what has not been done?

Unquestionably, many orthopaedic surgeons have given countless hours of time and extremely liberal sums of money to research in their own institutions. It is to their particular credit that this has been done. It is also to our credit that in 1955 the two major orthopaedic societies joined hands to organize the *Orthopaedic Research and Education Foundation* dedicated to the advancement of knowledge in our special field; this was a most significant forward step.

As important as the organization of the Foundation is the fact that 97 per cent of the more than 500 contributors to the Foundation have been *orthopaedic surgeons themselves* and in the first three years of the Foundation's existence, nearly $200,000.00 has been pledged and over $115,000.00 in cash has been received. With these funds thirty-four grants have been made in seventeen states, totaling approximately $80,000.00. Some of the research projects so financed have been completed and the results published. They comprise important contributions to medical knowledge.

Of this beginning, the orthopaedic profession can surely be proud. The funds have been raised without fanfare or publicity and without appealing to the public. It is the result of the efforts of a group of professional people who have been trying quietly to fulfill the responsibilities of their calling.

However, if the Foundation is to fulfill its purposes, developing comprehensive and sound programs in research and education, it must not only increase the number of research grants, but also improve, by means of grants, the orthopaedic educational programs in our medical schools. To do this the Trustees of the Foundation have set a minimum goal of $100,000.00 for the coming year and have laid plans to approach selected foundations and corporations with an interest in our specialty, in addition to the orthopaedic surgeons themselves. The success of this expanded and much-needed fund-raising activity inevitably depends on the active interest of the orthopaedic profession itself. At present less than one-fifth of the profession in the United States has contributed to the Foundation. It is imperative and only right that the Foundation should go forward with increased support from those whose life work is in orthopaedics.

Today we hear much of the need for good public relations. Good public relations means "earned recognition," with emphasis on the word *earned*. In the Foundation, the orthopaedic profession has the finest public relations weapon it could possibly find. Untold time and service have been spent to develop it. But unless orthopaedists themselves support their own creation, the work of the last few years will have been in vain.

On the other side of the public relations coin is the need to tell people about the good works already accomplished. Perhaps the Foundation has not adequately publicized its work within the specialty but be that as it may; *action*, both by the Foundation and by individual orthopaedic surgeons, is needed now or the "light may go out."

The Board of Trustees of the
Orthopaedic Research and Education
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Proceedings

LANKENAU HOSPITAL ANNUAL RESEARCH CONFERENCE

October 30 and 31, 1958

Bone As a Tissue was selected as the subject for discussion at the Annual Research Conference at the Lankenau Hospital, Philadelphia, October 30 and 31, 1958, because of its wide application to many fields of medicine. It was considered appropriate to review this field at this time in view of the recent significant advances in this area, which no doubt will markedly influence future trends in bone research.

More than 400 physicians and research workers from different areas of the United States attended this two-day conference in which sixteen invited speakers, among them six of the leading European workers, discussed the different aspects of bone growth and development.

The First Session was devoted to **osteoporosis:**

H. A. Sissons, M.D., Department of Pathology, Institute of Orthopaedics, London, England, discussed osteoporosis of Cushing's syndrome, describing the structural bone changes in Cushing's syndrome and comparing them with the effects of cortisone administration in experimental animals. The speaker's evidence from fourteen autopsy cases supported the earlier concept that in Cushing's syndrome there is a diminution of osteoblastic activity and absence of any obvious exaggeration of osteoclasis. Furthermore, his evidence suggested that in Cushing's syndrome the bones fail to respond to fractures in a normal manner.

Following this, H. A. Sissons discussed the general question of the recognition and assessment of the cellular processes responsible for the maintenance of normal bone structure and for the development of various forms of bone rarefaction. He outlined his ideas on the quantitative aspects of osteoblastic and osteoclastic activity as a morphological approach to the problems of bone turnover, based on the determination of the areas of the bone surface occupied by bone formation and bone destruction and the measurement of the rates of linear bone formation and destruction.

M. R. Urist, M.D., Division of Orthopaedics, Department of Surgery, Medical Center, University of California, reported on his observations bearing on the problem of osteoporosis, presenting a summary of clinical and laboratory studies of cases with fractures that required orthopaedic measures, as well as cases that were undiagnosed and asymptomatic, in an attempt to outline the broad scope of the problem of osteoporosis. He concluded that the principal etiological factor in the pathogenesis of osteoporosis is not known and that there is no correlation between dietary intake of calcium and osteoporosis. He focused attention on the possible relationship between osteoporosis and gastro-intestinal ulcers and discussed the evidence for and against the endocrine theory pointing out that the great majority of patients with osteoporosis have no specific endocrine disease. Preparations of undecalcified cortical bone, examined in cross-section by histological, microradiographic, and micro-autoradiographic techniques showed that in osteoporosis the bone resorption phase of Haversian remodeling appeared to have greatly accelerated and that the bone reconstruction phase was active but not sufficient to replace the loss of bone mass. Bone resorption in osteoporosis occurs almost entirely without the action of osteoclasts. He emphasized that osteoporosis of the spine and hips is becoming increasingly common on all orthopaedic services and should receive a proportionately greater amount of attention from orthopaedic surgeons. This disorder is largely responsible for the unalterable 30 per cent incidence of non-union in fractures of the hip, not because of inability of the bones to heal, but because osteoporosis causes crumbling of the fracture surfaces. For this reason, it is technically impossible to achieve good contact and internal fixation by metallic appliances in many cases. He recommended an examination of lateral roentgenograms of the spine in order to determine the presence or absence of osteoporosis and to avoid ineffectual methods of metallic internal fixation in many patients with fractured hips.

B. E. C. Nordin, M.D., Department of Medicine, University of Glasgow, Scotland, suggested that osteoporosis is the result rather than the cause of negative calcium balance. His argument rested upon the following points:

1. Calcium deficiency produces osteoporosis in animals;
2. In a majority of a series of patients with clinical osteoporosis, evidence was obtained of low calcium intake, malabsorption, or hypercalcuria;
3. The calcium accretion rate in a small number of patients was normal;
4. Most patients with osteoporosis can be brought into positive balance simply by feeding them calcium;

5. In the majority of patients, backache can be relieved or cured simply by feeding them calcium supplements;

6. There are theoretical grounds for believing that calcium deficiency will lead to osteoporosis;

7. It is known that some individuals are consuming very little calcium, and it is very possible that a proportion of these are in negative calcium balance;

8. It is probable that since 99 per cent of body calcium is in the skeleton, a reduction in skeletal mass would be due to mineral, rather than protein, deficiency.

G. Donald Whedon, M.D., Metabolic Disease Branch, National Institute of Arthritis and Metabolic Diseases, National Institutes of Health, reported on his comprehensive studies on the role of atrophy of disuse in the development of osteoporosis. He concluded from studies of the effects of disuse or immobilization in normal subjects, that the calcium losses which have been observed would, if continued, be expected to lead to the acute development of osteoporosis. In studies of paralytic poliomyelitis involving more complete immobilization, greater calcium losses were noted with the development of osteoporosis which was quantitatively related to calcium loss. The relationship of these greater calcium losses to disuse has raised questions as to the presence and importance of direct neural influences on bone. The speaker emphasized the paucity of our knowledge concerning disuse atrophy of bone.

In the discussions which followed these papers, Edward C. Reifenstein, M.D., The Squibb Institute for Medical Research, New Brunswick, New Jersey, outlined his hormonal theory of osteoporosis, emphasizing the antianabolic concept.

Dr. Urist stated that there are factors other than estrogen, androgen, and compound F in the blood that are responsible for osteoporosis, factors which he termed *antiosteoporosis factors*. He pointed out that the Reifenstein-Albright hypothesis of 1956 is unproved but that the bulk of evidence would probably be in favor of it. He voiced the need for a suitable laboratory animal in which osteoporosis can be produced and felt that the final solution of this problem must come from such animal experimentation.

Dr. Nordin stated, in response to a question, that the key to his concept of calcium deficiency as the cause of osteoporosis is that osteoporosis occurs in persons who fail to adjust to a low calcium intake. Because of the abundance of calcium in the skeleton, this process of a negative calcium balance will require twenty or thirty years to produce roentgenographic changes in the skeletons.

Dr. Urist pointed out, in contrast to this, that he found osteoporosis in persons who were taking over 800 milligrams of calcium a day. He pointed out that one of the essential points of difference between his views and those of Dr. Nordin was that he did not think, by placing an animal on a low calcium diet, one can produce what is seen clinically and is identified as osteoporosis. It was Dr. Urist's opinion that the low calcium diet produced what Aub and his group described as resorption of the readily available calcium deposits. What happens in man is something different. He considers osteoporosis to be a disease which affects not cancellous bone indiscriminately anywhere but primarily cortical-bone formation. He felt that a definite diagnosis of osteoporosis should be made only on the basis of a biopsy of cortical bone.

The Second Session, under the chairmanship of Cyril L. Comar, Ph.D., Laboratory of Radiation Biology, Cornell University, Ithaca, New York, was devoted to the **Dynamics of Calcium Metabolism:**

The first speaker, William F. Newman, Ph.D., Department of Pharmacology and Biochemistry, University of Rochester, New York, discussed the chemical dynamics of bone mineral. He pointed out that although the structure of the bone mineral is fairly well understood, there remain several very important unresolved problems. The exact nature of the carbonate in bone is unknown. The mechanism by which new crystals are formed in bone and cartilage is not understood. He noted that perhaps the most promising development in this area is the discovery that collagen fibers can nucleate crystal formation at concentrations below the point of spontaneous precipitation. There is a considerable body of evidence which indicates that the degree of mineralization increases with the age of the skeletal unit, but it is not yet established whether this involves continued growth of individual crystals or the formation of new ones. He emphasized that the nature of the water in bone is only partially understood, although it is clear that the skeleton as a whole is quite dehydrated. This dehydrated state may be responsible for the relative inertness and lack of physiological reactivity of bone. The gradual loss of water as skeletal units age may be responsible for the apparent irreversibility of the many ionic interchanges undergone by the skeletal mineral.

Göran C. H. Bauer, M.D., University of Lund, Sweden, discussed the kinetics of calcium and strontium metabolism. He concluded that animal experiments, in which isotope techniques have been used, have contributed to a better insight into normal processes such as bone growth and repair and into pathological conditions such as vitamin-D or protein deficiency.

Isidore Gersh, Ph.D., Department of Anatomy, University of Chicago, discussed the

dynamic aspects of ground substance and calcification. He reviewed the significant observations which form the basis for current concepts concerning the structure and nature of the ground substance and the origin of collagen fibrils in the ground substance. He described the procedure for producing suitable specimens for the study of the dynamics of ground substances, and analyzed the factors which influence the relative proportions of water-rich and colloid-rich phases in the ground substance. He pointed out that ground substance is organized as a system of discontinuous water-rich, colloid-poor submicroscopic vacuoles. These are separated by the denser water-poor, colloid-rich continuum. These two elements of the ground substances are considered to be the morphological expression of a two-phase system. The relative proportions of water-rich and colloid-rich components may be altered, by maturation and aging, by the administration of various hormonal substances, and, under certain conditions, by vitamin C. As the two phases are assumed to be in thermodynamic equilibrium, changes in the relative proportions of each phase will result in changes in the concentration of the ions in the ground substance. The colloid-rich phase seems to be the site in the ground substance where the collagen fibrils originate. He also suggested that normal osteoid can be regarded as a form of ground substance of connective tissue, and that it seemed probable that the findings on the factors influencing the physical and chemical lability of the ground substance of muscle, which are more readily subject to study, may be extended to osteoid. Dr. Gersh suggested that a large part of the answer to the question why tendons generally do not calcify normally, while bones do, will be found in the dynamics and transformations of the ground substance. He pointed out the possibility that the rate of formation and maturation of ground substances may be the deciding factor in determining whether a tissue becomes calcified. One hypothesis is that ground substance matures more slowly in tendon than in bone.

John H. Marshall, Ph.D., Radiological Physics Division, Argonne National Laboratory, Lemont, Illinois, discussed the problem of microscopic metabolism of calcium in bone, reporting the results of his studies in which he used the new techniques of quantitative autoradiography, microradiography, and computer analysis of blood and bone specific activities. He pointed out that recent experiments with radioactive tracers in dogs have revealed an important component of calcium metabolism in bone. It has been found that calcium, as well as the calcium-like isotopes Ca^{45}, Sr^{90}, and Ra^{226}, diffuse into and out of bone mineral at an appreciable rate even in regions of mature bone which have been thought to be metabolically inert. Called, more accurately, long-term exchange, this component of calcium metabolism accounts for as much calcium transfer between blood and bone in an adult animal as the well known components, accretion and resorption. However, because practically all bone crystallites are involved in this exchange, the time constant is long, so that a radioactive isotope deposited in bone is not returned to the blood by exchange to an important extent until several years after injection. The mechanism responsible for this component is probably exchange at crystallite surfaces, possibly coupled with intracrystalline diffusion. The existence of long-term exchange will modify our interpretation of accretion-rate measurements based on radioactive uptake and will increase our estimates of the availability of skeletal calcium to the circulating fluids.

The Third Session, in the morning of October 31, under the chairmanship of Friedrich Wassermann, M.D., Ph.D., Division of Biological and Medical Research, Argonne National Laboratory, Lemont, Illinois, was devoted to **Ultrastructure of Bone.**

Sylvia Fitton Jackson, M.D., from the Biophysics Research Unit, King's College, London, England, presented her studies on fibrogenesis and matrix formation. She pointed out that the processes involved in fibrogenesis and the formation of matrix are numerous and many may as yet be unknown. Seven successive steps, however, which are probably concerned in the production and aggregation of the collagen macromolecules, have been defined. These steps may be regarded as the skeleton of the processes which take place in the tissues during fibril formation. Both *in vivo* and *in vitro* experiments have supplied data which have been used to build up a more coherent picture of the possible mechanisms involved. The defined steps in fibril formation may be regarded as distinct individual reactions. It must be stressed that the problems and mechanisms involved have been purposely stated in their simplest terms and that most of the reactions in the successive steps take place concurrently with one another. It is clear that more information is required before any of the mechanisms involved in these steps in tissue fibrogenesis are understood and established as correct or discarded. The examination of the fine structure and chemical composition of periosteal bone and of the four regions of the growth cartilage in long-bone rudiments has supplied data at different histogenetic stages and demonstrated certain variations which may be concerned in the mineralization of the tissue. The morphological development of apatite in bone and cartilage shows differences in formation which may be related to the method of initial seeding of the apatite crystals in relation to the component collagen fibrils.

Robert A. Robinson, M.D., Department of Orthopaedics, The Johns Hopkins Hospital,

Baltimore, Maryland, pointed out that a specimen of bone cleaned of periosteum and medullary marrow can be divided into two major parts: (1) the marrow-vascular-osteocyte space and (2) the calcified bone matrix. These two parts are intertwined so intimately that the chemical analysis of bone is quite confusing unless they can be separated. The chemical analysis of bone must be correlated with the anatomy of the bone. This concept is necessary in evaluating the chemical analysis of porotic bone. Considerations regarding the marrow-vascular-osteocyte space lead the author to believe that a rather constant analysis pattern can be derived for the fat-free marrow. Electron-micrographic investigations when considered in conjunction with results of other investigative techniques demonstrate both a standard pattern for calcified osteoid and variations from this standard in respect to percentage calcification. If the standard marrow-vascular-osteocyte pattern is combined with the standard calcified osteoid pattern and its several variations, an intelligent estimate of the chemical composition of a specimen of bone can be made within the perimeters of its density both when hydrated and when dry. The over-all results of chemical analyses of bone in relation to its anatomy were shown in tables and graphs. Although these standard analysis patterns were based on studies on dog bone, the author believed that they can be applied equally well to human bone. Human bone in general has a slightly lower hydroxyapatite to collagen ratio than does dog bone. Otherwise the component parts of these two organic matrices and their marrow apparently have similar mass to volume or density characteristics.

Arne Engstrom, M.D., Department of Medical Physics, Karolinska Institute, Stockholm, Sweden, reported on his studies related to the ultrastructure of bone mineral. He mentioned that the nature of the mineral fraction of bone has been discussed extensively since the early work of deLong in 1926. At that time it was considered that the bone mineral was an apatite, but due to the confusion caused by the work of D. McConnel, concepts such as carbonate apatites were introduced. McConnel based his conclusions on results from the study of some mineral apatites. From the work of D. Carlstrom, at the Department of Medical Physics, Karolinska Institute, it is now clear that the mineral carbonate apatites are microcrystalline compounds with hydroxyapatite crystals in an amorphous matrix of carbonate. It is also well established that bone apatite occurs as microcrystals which are so small that a considerable fraction of the atomic groupings is in a surface position. It is this small crystallite size which has caused the diverse interpretations of the structure of the bone salt. Bone salt has unit cell dimensions of $a=9.42\pm0.03$ Å and $c=6.88\pm0.01$ Å, while the dimensions of pure synthetic hydroxyapatite are $a=9.421$ Å and $c=6.882$ Å.

The size of bone crystallites has been measured by studying the diffraction profiles of the wide angle x-ray lines, the intensity distribution in the low angle particle x-ray scatter, and by electron-microscopy. The results of these investigations can be summarized in the following way. The bone crystallites are about 20 to 40 Å in diameter and 600 Å or more in length with a kinked structure. The important point is that the crystallites have such small dimensions that 20 to 50 per cent of the groups are in a surface position, thus in effect the crystallites act as an effective ionic exchanger. In fact the surface of the bone crystallites can be considered as a garbage can for all sorts of ions. This is one way in which the bone salt plays a role in the homeostatic regulation of ions.

Crystallographic investigations have shown that when animals are fed strontium very little of this is substituted in the lattice (for example, into the columnar calcium ions) as evidenced by changes in the lattice parameter as deduced from the x-ray diffraction patterns. Most of the strontium is surface bound. The study of bone apatite would appear to be a problem of surface chemistry.

The elongated bone crystallites are arranged along the collagen fibers, but it is not known whether there is any specific linkage between the apatite and the collagen. As other types of calcifications occurring in the absence of collagen show the same types of crystallites, the typical arrangement in bone seems to be directed mainly by physical forces.

Finally, Pierre Lacroix, M.D., Institute of Anatomy, University of Louvain, Belgium, presented the results of his investigations with autoradiography. Ca^{45} autoradiographs of adult and growing bone were analyzed with particular reference to the morphological changes during calcification of bone matrix as revealed by microradiography combined with routine histological methods. His presentation was based on a series of photomicrograms and cannot, therefore, conveniently be presented in abstract form.

The Final Session, under the chairmanship of John E. Howard, M.D., Department of Medicine, The Johns Hopkins Hospital, Baltimore, Maryland, dealt with **vitamin D, parathyroid, citric acid, calcium, and phosphorus:**

D. Harold Copp, M.D., Department of Physiology, University of British Columbia, Vancouver, Canada, discussed the role of the parathyroid in the homeostasis of blood calcium. He outlined his theory for the mechanism responsible for the very precise regulation of the plasma calcium level. He pointed out that the plasma calcium exists in equilibrium with that in interstitial fluid. The plasma receives its calcium from intestinal absorption and from bone resorption. Calcium is lost from the plasma by excretion in urine and faeces and by deposition in bone. Possible changes in plasma calcium level are buffered by the labile storage pool in

bone, which may correspond to the calcium in the hydration layer and surfaces of the crystals of bone salt. The maintenance of a normal level of plasma calcium depends on the activity of the parathyroid glands which probably act by a feed-back mechanism as suggested by McLean. Hypocalcaemia apparently stimulates the parathyroid glands to produce their hormone which in turn promotes mobilization of calcium from bone. It is probable that hypercalcaemia inhibits the parathyroids. There is considerable evidence that parathyroid extract increases the activity of bone cells, particularly with respect to citrate. Neuman and his associates suggest that the citrate ion increases the solubility of bone salt and thus promotes calcium mobilization. It may also increase the activity of calcium in the labile bone storage pool.

Because the parathyroidectomized animal can mobilize calcium from its skeleton and can maintain the blood calcium, even though at a level much below normal, it seems likely that the hormone acts to increase the activity of a mechanism already present. In this respect, it would be analogous to the thyroid hormone, which is not essential for metabolic activity but is required to increase metabolism to its normal optimal level.

Harold E. Harrison, M.D., Department of Pediatrics, Baltimore City Hospital, Baltimore, Maryland, discussed the physiology of vitamin D. He pointed out that since cortisone blocks the citrate-increasing action of vitamin D without preventing its essential antirachitic activity, the citrate effect does not appear to be the primary biochemical mechanism of vitamin D action. Nevertheless, the citrate phenomenon may be a clue to the nature of vitamin D action since the movement of citrate is possibly related to the transport of ions across cell membranes. Vitamin D appears to influence the rate of movement of calcium and phosphate ions through the intestinal mucosa and the renal tubular cells as well as influencing the movement of calcium between the solid phase of bone salt and the liquid phase of the body fluids. In certain of these functions an interaction of vitamin D and parathyroid hormone seems to exist. Elucidation of vitamin D action and explanation of parathyroid hormone effect are probably mutually dependent.

Smith Freeman, M.D., Ph.D., Department of Biochemistry, Northwestern University Medical School, Chicago, Illinois, discussed citrate metabolism with special reference to calcium and bone and the possible role of citric acid in mobilizing calcium from the skeleton. During recent years it has been demonstrated that citric acid is widely distributed in calcareous deposits formed in living organisms. The highest concentration in the animal body is found in the skeleton. Citric acid is continually entering and leaving the blood stream. There is some evidence to indicate that the skeleton may be the site of origin of at least part of the citric acid contained in the blood. Citric acid is being removed continuously from the circulation and metabolized by the kidneys. The physical and chemical natures of citrate and calcium make certain reactions between these two substances inevitable. Both vitamin D and parathyroid function appear to influence the concentration of citric acid in the blood stream. There is presumptive evidence to suggest that citrate may play a role in the normal homeostatic mechanisms by which calcium ion concentration is controlled in the animal body. The enzymatic and cellular mechanisms by which citrate metabolism is related to that of calcium are not understood.

Franklin C. McLean, M.D., Department of Physiology, University of Chicago, concluded the conference with a brilliant summary of phosphorus metabolism, and stated: "Perhaps we can go no further at this time than to suggest that there is now reason to believe that phosphate, heretofore regarded as the passive member of the calcium-phosphorus team in calcification, may be found to provide such activity or energy as may be necessary for this process, or more particularly for its initiation or seeding. In this case the passive or secondary role may have to be assigned to calcium."

Book Reviews

THE MANAGEMENT OF FRACTURES AND DISLOCATIONS. An Atlas. Volumes I and II. Anthony F. DePalma, M.D. Philadelphia, W. B. Saunders Company, 1959. $35.00.

This two-volume atlas is the author's answer to the need for a ready and rapid reference on the treatment of a specific injury. Dr. DePalma has outlined the pertinent factors in the management of any given fracture or dislocation in a simple and abbreviated text, illustrated with pen line sketches. The pertinent steps only are demonstrated. Having undertaken this approach to his task, he presents the method which he personally feels is the most satisfactory. The reader is not left any choice in the management of a given injury. The fracture or dislocation is clearly described, and its treatment is carefully outlined, step by step. The methods are dogmatic, but the material is clear and concise. Each individual injury is presented as a basic unit, and completed through the rehabilitative phase. Unfortunately, this approach calls for considerable reduplication of printed material and illustrations. For example, the type and position of the Minerva jacket is demonstrated nine different times during the section on cervical-spine injuries. Space and printing could be reduced by this seemingly unnecessary repetition.

Certain sections of these two volumes overemphasize the simple conditions, and yet do not furnish enough detail for the more complicated problems. This is typically true in *injuries of the thoracic cage*, where twelve pages are devoted to the management of uncomplicated fractures of the ribs and sternum, and yet only one page is devoted to open *pneumothorax* and its treatment.

Particularly valuable are the clear sketches of the actual steps of reduction in various fractures. Here the inexperienced quickly can find, well illustrated, the method of reducing fractures and the type of cast recommended. The volumes are large and cumbersome. No attempt has been made to provide references. The two volumes should certainly be available to all who are called upon to treat frequently and infrequently trauma to the musculoskeletal system. Every hospital would do well to have the volumes available in their emergency facility for ready reference, as they provide a quick, compact discussion of a given injury, and present acceptable methods of treatment.

William R. MacAusland, Jr., M.D.

RECENT ADVANCES IN CEREBRAL PALSY. Edited by R. S. Illingworth. Boston, Little, Brown and Company, 1958. $12.00.

Recent Advances in Cerebral Palsy presents a good discussion of all aspects of cerebral palsy by contributors who are leaders in their respective fields. It consists of an up-to-date summary of the literature on the subject, both orthopaedic and otherwise. Inasmuch as the patient with cerebral palsy is ordinarily best treated by a team of individuals including an orthopaedist, pediatrician, neurologist, physical therapist, speech therapist, psychologist, and neurosurgeon, this book is written with this approach in mind. Except for the three American contributors to this text of fifteen chapters, the title might well be *Recent Advances in Cerebral Palsy in Great Britain*. It is only fair to say, however, that there are many references to the American literature on the subject, and there are excellent chapters by Dr. Pearlstein, Dr. Phelps, and Dr. Meyers. The various techniques of treatment of cerebral palsy might have been discussed in more detail since there is so much controversy about these.

There is an excellent chapter devoted entirely to the role of orthopaedic surgery in the treatment of cerebral palsy. Some controversial points are mentioned. Tibial tubercle transplants are described, but these should not be done during the growing period because of the danger of disturbing the growth of the epiphysis here.

This textbook is well written, and it will prove to be an excellent reference volume. It should be read by all who are engaged in the treatment of patients with cerebral palsy.

Henry H. Banks, M.D.

TREATMENT OF CANCER AND ALLIED DISEASES. Ed. 2. Vol. 1: Principles of Treatment. George T. Pack, M.D., and Irving M. Ariel, M.D. New York, Hoeber-Harper, 1958. $22.50.

This is the first volume of a projected nine-volume series modernizing the previous edition published in 1940. In the interval of two decades increased clinical and research emphasis on the cancer problem has broadened our knowledge extensively, and this initial volume of the new series reflects this added knowledge. Of the original twenty-five authors who wrote this section of the treatise, only four have contributed to the new edition. Three have completely rewritten their contributions and only the work of one—Broders on grading of tumors—has remained essentially unchanged. In my opinion this too should have been rewritten because of the widespread differences of opinion on the value of a grading procedure in different tumors not considered in the presentation. The new edition, in this section at least, is double the size of the old, and is the work of twice the number of contributors.

The series will have the same framework as in the previous edition, that is, each volume after the first will consider tumors of anatomical regions of the body. The first volume discusses a variety of topics pertinent in a general way to the subject, but not to any specific clinical entity. The organization of this volume is admirable—it begins with cancer prevention and passes through methods of diagnosis and pathology, principles of surgery, radiation, and chemotherapy, and ends with a chapter on the reporting of end results of cancer treatment.

For those interested in orthopaedic surgery, each of these chapters has material which should be understood by the orthopaedic member of a cancer therapy team. He will benefit by increased familiarity with several sociomedical subjects which ordinarily would not come to his notice—the carcinogenic agents which may be encountered as environmental or occupational hazards, the organization of clinics, home-care facilities, and arrangements for terminal and nursing care. In an area somewhat closer to his direct responsibilities for the care of patients, a general review of the principles of radiation physics and biology and the specific uses in therapy of radioisotopes will be appreciated by many readers. Similarly, the fields of chemotherapy and hormone therapy are well summarized for the practitioner, but one wonders how many years will elapse before most of the agents considered will be obsolete.

Naturally, the orthopaedic surgeon will be most concerned with those chapters which relate specifically to the surgeon's handling of the patient. It is doubtful whether any surgeon, on the basis of material presented in this book, would feel himself competent to direct the care of a cancer patient with regard to radiation, chemotherapy, or the like. However, as a member of a team, his surgical responsibilities remain paramount. It is to be expected that the local surgical problems will be considered in the appropriate subsequent volumes. In the present one, the general principles of surgical preoperative and postoperative care, fluids, estimation of risk, and preparation of poor-risk patients, occupy a good portion of this section of the book. Since these considerations are pertinent to treatment of any surgical patient, one wonders whether they should be included in a volume of this nature. The remainder of the section, devoted to electrosurgery and to vascular surgery, as it pertains to the treatment of cancer, certainly are pertinent.

The work of many authors is necessarily uneven, and this volume covers in summary fashion much material in diverse fields. As such, it presents a variably fitting introduction to the more specific volumes to come. Faults of complete omission of topics are few indeed —perhaps the experimental field of tumorigenesis, immunology, transplantation, and the like should have been included. The book may be recommended for a modern summary of the subjects mentioned above, treated in a simple understandable way suitable for all medical practitioners and students, but not suitable for study in depth, or for reference.

Jonathan Cohen, M.D.

Handbook of Orthopaedic Surgery. Ed. 5. A. R. Shands, Jr., M.D. St. Louis, The C. V. Mosby Company, 1957. $9.75.

The Fifth Edition of this compact volume meets well the author's aim—to "assist in the teaching of orthopaedic surgery". Dr. Shands suggests the book for medical students, general practitioners, nurses, and therapists and at the same time offers a great deal for residents and practicing orthopaedists. He defines clearly the area to be considered—orthopaedic surgery exclusive of fresh fractures, plaster technique, surgical technique, and orthopaedic apparatus.

Within this framework the material is clearly organized and well presented. The salient features of each subject are succinctly put. For detailed or exhaustive information on a topic, the reader must use liberally the excellent bibliography. Attention is drawn to both sides of controversial points and then the author's preference is given. The illustrations are apt, appropriate, and abundant. The sections dealing with pediatric orthopaedics are stronger than those dealing with adult problems. The Handbook is strong on definitions. It also contains an interesting amount of the history of orthopaedics.

One wonders about certain statements such as: "fibrous dysplasia is almost always unilateral"; "the cases most favorable for hip arthroplasty are those in which the ankylosis has been the result of . . . an acute pyogenic infection"; "currettage of tuberculosis sinuses is nearly always futile"; and "Reiter's syndrome is a variant of rheumatoid arthritis". However, to challenge only these statements in a 600-page book is high praise.

William H. Harris, M.D.

Third Tissue Homotransplantation Conference. Annals of the New York Academy of Sciences, Vol. 73, Art. 3, pages 539-868. $5.00.

This series of papers is the result of the Third Tissue Homotransplantation Conference held by the New York Academy of Sciences, Section of Biology, February 6 and 7, 1958. Two preceding volumes, consisting of the papers presented at the First (1954) and Second (1956) Tissue Homotransplantation Conferences have been published.

This volume is divided into four parts. Part I concerns "Embryonal, Fetal, Neonatal,

and Infant Tissue Transplantation". Although this section is largely concerned with the phenomenon of take of homografts of embryonal tissue in experimental animals, Peer and his associates reported that in humans, skin grafts from mother to son and daughter survive longer (although ultimately rejected) than from father to son and daughter. Part II considers the "Immunogenetics of Tissue Transplantation", and Favour points out that "in invertebrates who can be successfully homografted, the plasma cell system is absent, and the capacity to make gamma-type serum globulin is vestigial". Berrian and Brent reported further experimental evidence that "antibody-like reactive sites are created on or within the lymphoid cells" in skin homotransplantation, and Scothorne noted that "although not finally proved, it seems likely that the antigens of the graft reach the antibody forming apparatus of the host directly via the host lymphatics in the graft bed, and that immunization of the host can occur in the absence of an intrinsic lymphatic drainage of the graft". Part III consists of papers on "Antibodies and Antigens in Tissue Transplantation". Stetson and Demopoulos, and Gorer, reported on the controversial subject of the ability of immune sera passively to transfer specific homograft immunity. Converse and his associates, in answer to the question "How soon after transplantation of a skin homograft does transplantation immunity develop?", state that "the establishment of the vascularization of a skin homograft is required for the development of transplantation immunity. . . . The presence of the skin homograft in the host bed for a period of about 48 hours is required". Part IV concerns the "Graft-Versus-Host Reaction and Acquired Tolerance in Tissue Homotransplantation". Several papers considered the phenomenon of the effect of the graft against the host, as in "runt disease" (in new-born mice inoculated with adult homologous spleen cells), and "homologous disease" (attributed to the immunological reaction of successfully grafted homologous lymphoid elements or their bone marrow progenitors against the foreign tissue antigens of the toleraat host). Kamrin reported that treatment of rats with the alpha-globulin fraction of rat serum resulted in a survival rate of 60 per cent in parabiotic rats, as compared to 25 per cent survival in non-treated rats.

Only a few of the papers in this volume have been mentioned above, but there are many more that warrant close study. Unfortunately, there is nothing in this volume specifically concerning bone homografting, but a knowledge of the current investigations in all phases of tissue homografting is essential to anyone interested in experimental bone-grafting and transplantation.

Paul H. Curtiss, Jr.

Manual of Upper Extremity Prosthetics. Ed. 2. Edited by William R. Santschi. Los Angeles, Department of Engineering, University of California at Los Angeles, 1958.

This is a revised and larger edition of Upper Extremity Prosthetics compiled by the Department of Engineering, University of California at Los Angeles. It is a practical handbook encompassing material pertinent to the functions of each member of the prosthetic clinic team. The value of a team approach to prosthetics is emphasized; surgeon, prosthetist, and physiotherapist. Following in logical order are the detailed instructions for prescription of a limb, measurements and fabrication, and training of the amputee, with a generous number of clear sketches and photographs.

Engineering principles and component parts of the devices are described and pictured in detail. While for a surgeon an intimate knowledge of these is perhaps not essential, it increases considerably his ability to head the clinic team. Materials and processing for plastic laminate construction of sockets are shown in detail and are of primary value to the prosthetist. Physiotherapists will find particular interest in discussion of preprosthetic stump conditioning, details of harness and control systems, and the procedure to follow in training a patient to use his limb with maximum efficiency.

To my knowledge, the material embodied in this manual will enable the clinic team to give the patient the most functional and cosmetically desirable plastic laminate prosthesis. While engineers continue to apply their special skills to the problem of prosthetics, and improved materials and techniques are constantly evolving, it may be expected that prosthetics will be a dynamic area of endeavor for the interested surgeon.

Richard A. Mayo, M.D.

Pathology for the Physician. Ed. 6. William Boyd, M.D. Philadelphia, Lea and Febiger, 1958. $17.50.

From such a scholar, one could ask for little more than this delightful book. The history of medicine, the semantics of the jargon in pathology, and the anatomy of the several diseases are woven into a single cloth which, although often familiar, has many new patterns.

Each chapter is introduced with not only a historical setting but also with a useful review of normal anatomy and physiological function of the organ system. Personal concepts of function might be illustrated by his discussion of the homeostatic and excretory functions of the kidney wherein he states, "The excretory view is epitomized in the saying of the noseless story-teller of Baghdad: 'What is man, when you come to think upon him, but a minutely set, ingenious machine for turning, with infinite artfulness, the red wine

of Shiraz into urine?'" The more serious discussion of renal function that follows this statement is excellent.

The internist, for whom this book is designed, may find little need to review the eight pages devoted to the anatomical changes in typhoid fever but will enjoy the full discussion of the carcinoid syndrome, the Zollinger-Ellison syndrome, and alveolar proteinosis.

The excellent and comprehensive chapters on heart diseases and renal diseases are surpassed by a beautiful discussion of Diseases of the Nervous System. In this chapter, as throughout the book, the illustrations are excellent. A chapter devoted to the Internal Environment is only a fifteen-page presentation of a very complex subject and, although the author extols the usefulness of expressing electrolyte concentrations in milliequivalents per liter, he slipped several pages earlier and referred to the drop in serum potassium in familial periodic paralysis as "a drop in the serum potassium level from a normal of 18 to 22 mg. per 100 cc. to as low as 5 mg.". The chapters on Diseases of the Bones (27 pages) and Diseases of the Joints (25 pages) concern only medical problems and would not likely add much to the knowledge of the average orthopaedist.

Several presentations on controversial subjects may represent a minority opinion. He defends status thymicolymphaticus as an entity; he believes that the etiology of rheumatoid arthritis has as a "basic factor . . . a chronic or recurring infection, but the infecting agent has not been isolated"; and because he accepts multiple histological types of thymic neoplasms as separate ontogenetic tumors, must conclude that "one difficulty is that there is a great variety of thymic tumors, most of which are not related to myasthenia gravis".

The conclusions in several chapters reflect the author's respect for the unknown: on liver function he states ". . . with regard to its behavior in disease we are tempted to echo the famous remark of Fascatorius regarding the action of the heart that 'it was only to be comprehended by God'"; regarding the stomach, "the stomach, then, is a veritable sounding board for the emotions, and when one considers the bombardment it suffers from neurogenic, secretory and hormonal stimuli, not to mention exogenous irritants of every sort and description, the wonder is that anyone has a healthy digestion"; and on ulcers, he concludes that, "worry and nervous strain are associated with hyperacidity, perhaps explaining the prevalence of ulcer among surgeons and its absence in pathologists. I have known only one pathologist to suffer from a peptic ulcer, and he was a dean." The concluding remarks in the book are:

"Strange that a harp with so many strings
Should stay in tune so long."

The occasional disharmonies are presented very well in this book.

H. Robert Dudley, M.D.

Transplantable and Transmissible Tumors of Animals (Atlas of Tumor Pathology, Section XII—Fascicle 40). Harold L. Stewart, M.D., Katherine C. Snell, M.D., Lucia J. Dunham, M.D., and Samuel M. Schlyen, M.D. Washington, D. C., Armed Forces Institute of Pathology, 1959.

This is another Fascicle in the Atlas of Tumor Pathology of the Armed Forces Institute of Pathology. It contains a selection of fifty tumors of animals which are of frequent use experimentally. The transplantable tumors are those which require implantation of one or more cells in the host while the transmissible ones may be grown in animals after injection of cell-free extracts. The tumors were selected as representative samples of the variety of transplantable tumors which occur spontaneously or which may be easily induced in different animal species. Thirty tumors of mice, ten of rats, three of rabbits, two each of dogs and chickens, and one each of hamster, guinea pig, and frog are included. Each tumor is described in summary fashion and magnificently illustrated, often in color. While it is evident that this is not a complete listing of all of the experimental tumors of animals now being studied, this collection nevertheless has a good deal of value. It cannot be used as a complete reference work in this field, and thereby earn the title of atlas, but it can be used by laboratory workers to familiarize themselves with the common appearance of those tumors included and for reference purposes to the bibliography. The bibliography is by no means complete and often is inadequate and not up to date. The work is not meant for ordinary reading and serves only the limited purposes of reference for workers in experimental tumorigenesis and other phases of cancer research.

Jonathan Cohen, M.D.

Principles of Radiographic Exposures and Processing. Arthur W. Fuchs. Springfield, Illinois, Charles C. Thomas, 1958. $10.50.

Over the years, countless radiologists and x-ray technicians have been able to benefit from personal contact with the author as he solved their problems of radiographic technique. This volume is the summation of the many years of experience the author has had with the principles of radiographic exposures and processing. Part of the text has appeared previously in United States Army training manuals and part is directly from the author's lecture material.

The function of focus-film distance, the function of kilovoltage, and the function of milliampere-seconds in the formation of the radiographic image are just a few of the subjects covered. Following theoretical discussions of such topics as secondary radiation fog and "Heel" effect, experiments and demonstrations are given to illustrate their application. Extensive exposure tables for various routine projections are given to aid in setting up techniques in a new office.

The last third of this volume deals with processing procedures. Excellent examples illustrate ways in which processing errors produce inferior radiographs. Suggestions for correction of these faults are given.

This volume will be a valuable addition to every radiology office; the information it contains should be continually available to the radiologist as well as the x-ray technician.

Henry P. Pendergrass, M.D.

PICTORIAL HANDBOOK OF FRACTURE TREATMENT. Ed. 4. Edward L. Compere, M.D., F.A.C.S., Sam W. Banks, M.D., F.A.C.S., and Clinton L. Compere, M.D., F.A.C.S. Chicago, Year Book Publishers, Inc., 1958. $7.50.

The general practitioner who treats fractures occasionally, as well as students, will find the Fourth Edition of the *Pictorial Handbook of Fracture Treatment* as valuable and as easily understood a reference book as its predecessors. Theoretical discussion is kept to a minimum, necessarily, and widely accepted and practical methods of treatment are both clearly explained and illustrated by line drawings and roentgenograms. A further contribution to the clarity is the general use of anatomical terms in the description of fractures, a policy which is followed throughout the book.

The common fractures, such as those about the wrist and hand and the foot and ankle are described in considerable detail. At the same time the student is given the benefit of clear drawings illustrating the several techniques of hip reconstruction, many of early vintage. A description is also given of the use of the Soutter traction device and some of the elaborate techniques in the treatment of fractures of the calcaneus which do not enjoy a wide use at the present time. An especially valuable feature are the signposts indicating situations in which trouble might arise and the need for obtaining further consultation and advice.

There are a few techniques with which one might disagree, for example, the use of adhesive tape in the post-reduction fixation of elbow dislocation, together with a collar-and-cuff sling. One might question the necessity and effectiveness of marked dorsiflexion of the ankle in attempting to reduce small fragments of the posterior lip of the tibia. These, however, are small points and serve only to illustrate how hard one must look to find hairs to split. It is indeed interesting to note that the primary insertion of a prosthesis in the treatment of fresh subcapital fracture of the femoral neck is accepted, provided it is done in poor-risk patients whose life expectancy is no more than five years. One has the feeling that the reservations will be dropped in the next edition.

This little book is easy to read and easy to fit into a good-sized pocket. It is certain to compare favorably with its larger brothers and to retain its deserved popularity.

Richard M. Kilfoyle, M.D.

VASCULAR SURGERY. Geza de Takats, M.D., M.S., F.A.C.S. Philadelphia, W. B. Saunders Company, 1959. $17.50.

Extensively documented, this text on vascular surgery is nevertheless a personal book, frankly presenting the author's biases and opinions. His extensive experience and critical acumen make this a valuable attribute. Although the work has its weak chapters and some of the conclusions on therapy will be disputed by other vascular surgeons, in general the practical surgical principles and generalization which it presents and supports are sound, realistic, and valid in the light of current experience.

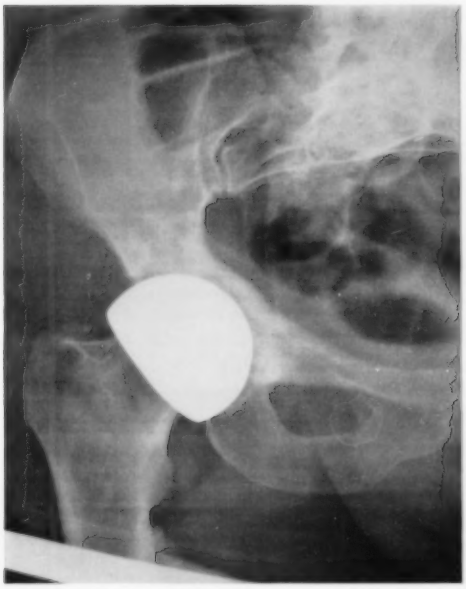
The book consists of sections on fundamental principles of vascular surgery, diagnostic methods, vascular syndromes requiring surgical care, and a final section on surgical technique. Consideration of the physics of circulation, in the first portion, is rather shallow, and occasionally, as in the case of statements about the *non-wettability* of endothelium, academically unfortunate. Despite this, fundamental observations on physiology and their practical importance are well brought home.

The section on diagnostic methods is greatly enriched by the author's experience, and will be useful to those wishing to develop their clinical ability in handling vascular disease.

The book's greatest value lies in the section on vascular syndromes which consists of thirteen chapters on the genesis, pathological anatomy, diagnosis, and treatment of the various conditions requiring the care of a vascular surgeon. This is not an extensive description of the use of arterial grafts and, although this aspect of the subject is covered, the author's experience is quite small by the current Texas standard. The section's strength lies in its handling of such entities as lymphoedema, cold injury, erythromelalgia, and connective-tissue disease, conditions rather neglected in the vascular surgeon's present enthusiasm for bigger and more spectacular reconstructive surgical procedures. These chapters make the book a most desirable reference text for the practicing surgeon.

Robert S. Shaw, M.D.

Thinning of hip joint cartilage, marginal spurring and cystic changes indicate osteoarthritis.



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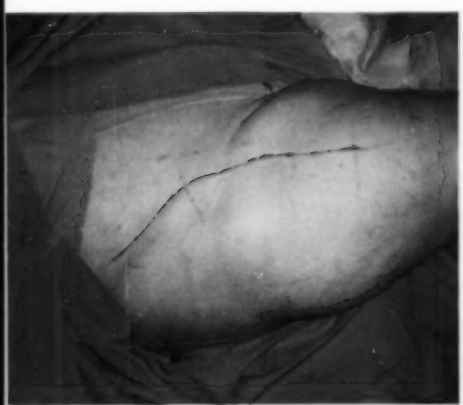

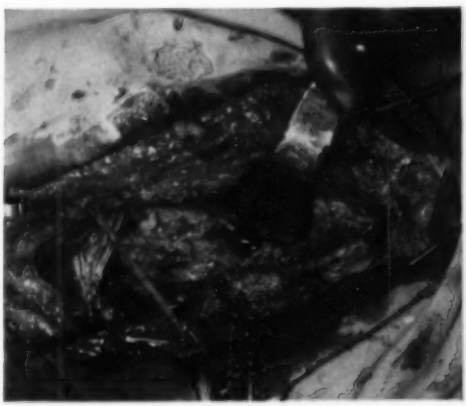

1. *(Illustration at left)* Anterior iliofemoral incision. Gloved hand is on medial side. **2.** *(Right)* Joint is now exposed. See how clearly color brings out the details: anterior capsule at bottom of wound; iliac crest at top; lateral femoral cutaneous nerve crossing below retractors.

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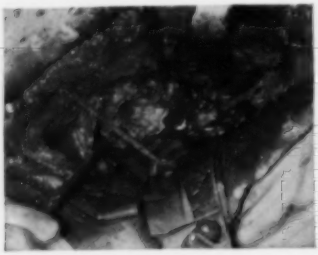

3. Capsule opened shows synovitis and irregularity of the osteocartilaginous junction of the head and neck.

4. Head of femur has now been dislocated. Note erosion of cartilage in the acetabulum.

5. Acetabular reamer positioned for cleaning cartilage from acetabulum. *Here color differentiates textures.*

6. Head of femur is being cleaned of all sclerotic and cystic substandard bone and degenerated cartilage.

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7. Shows cup on head of femur.

8. Hip, reduced, back in position prior to closing incision.

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GOLDSTEIN MONOGRAPH PUBLISHED AT OPPORTUNE MOMENT

The long search for authoritarian information on care of the scoliotic child has ended. Based on personal experiences over a twenty-year period, Dr. Goldstein's monograph, **The Surgical Treatment of Scoliosis,** moves into a vacuum of information in the orthopedic educational informative fields.

Dr. Louis Goldstein, *well-known Professor of Orthopedic Surgery at the University of Rochester Medical Center in New York,* has ELIMINATED ALL VARIABLES IN THE EVALUATION OF END RESULTS. Thirty-one cases are illustrated — all treated by one method and followed up by the author. He has emphasized important points in the discussion by preoperative and postoperative roentgenograms and clinical photographs.

The author's experience is an outstanding example of selective application. Every physician who deals with the problem of scoliosis should share in this summation of experience by an outstanding surgeon and his team of associates. *Copies of this latest monograph in American Lectures in Orthopedic Surgery available to the profession in June.*

Surgeons Welcome Two Recent Publications on the Hand

In view of present concern about effects of radiation in the production of mutation, it was both timely and important that **Professor Arthur J. Barsky's** work on **CONGENITAL ANOMALIES OF THE HAND AND THEIR SURGICAL TREATMENT** be completed in 1958. It is a statement of present day experience by a top flight plastic surgeon and includes for the busy practitioner step-by-step treatment of each condition (*copies still available at $5.75*).

Rounding out coverage of the hand, January 1959 saw the completion of **THE HAND: ITS ANATOMY AND DISEASES** by a Professor of Surgery at the Boston University School of Medicine — **John J. Byrne.** Emphasizing important details with unusually clear pen and ink drawings, Professor Byrne covers structure and development of the hand, infections, injuries, diseases, and reconstructive surgery (*408 pp., 233 il., $10.50*).

Authorities Clarify Two Difficult Entities

A late 1958 Second Edition of **Ruth Jackson's THE CERVICAL SYNDROME** (*205 pp., 215 il., $6.50*) presents the author's personal experiences with a difficult entity. New materials in this edition include 40 new illustrations and 1000 additional cases. A lot about baffling whiplash injury significances and x-ray findings as diagnostic aids. Useful in checking your own findings with your patients who have arm, shoulder and neck pains, or neck pain associated with headache!

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"Is It Rheumatism Doctor?"

So reads the subtitle of a brand new (March 1959) little monograph by **W. H. Gervis** on **ORTHOPEDICS IN GENERAL PRACTICE.** For "the little things that try us" Dr. Gervis offers from a life-time of experience successful methods and procedures of diagnosis and treatment. *Look over an on-approval copy at your leisure* (*$3.00*).

Detail Cut to a Minimum

For the whole orthopedic team Charles C Thomas, Publisher, has imported from England a limited number of **T. T. Stamm's A GUIDE TO ORTHOPEDICS** (*124 pp., 19 il., $3.00*). An ideal handbook on the art of maintaining functions of the locomotor system.

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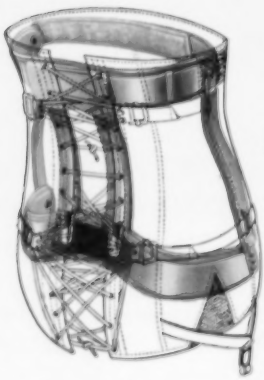

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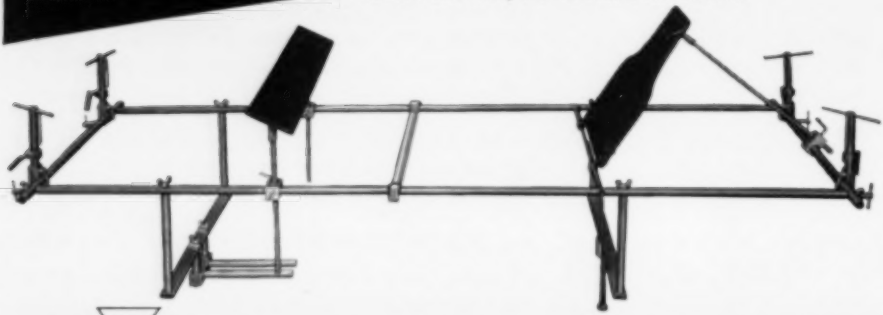

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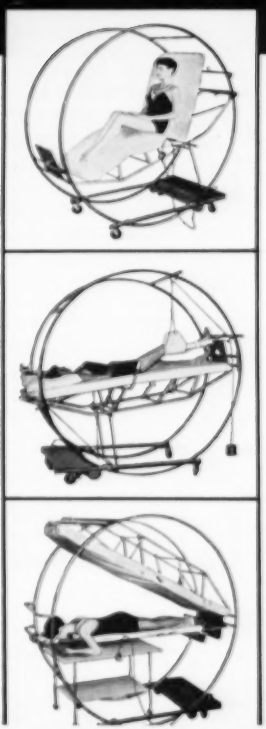

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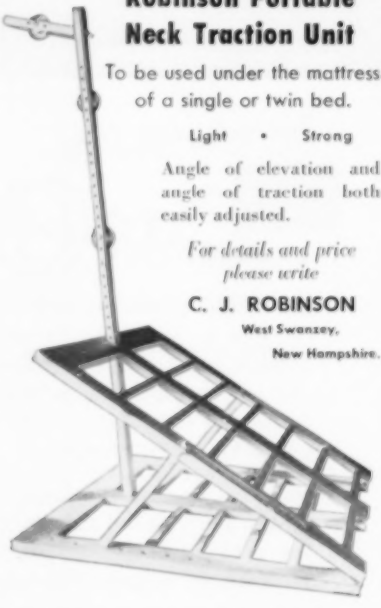

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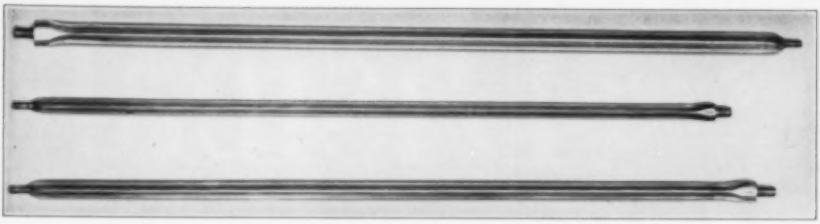

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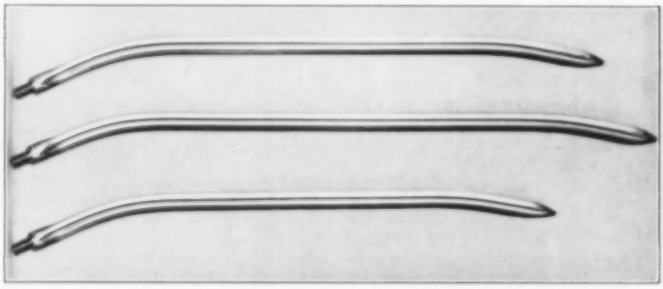

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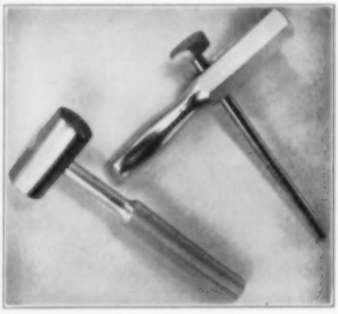

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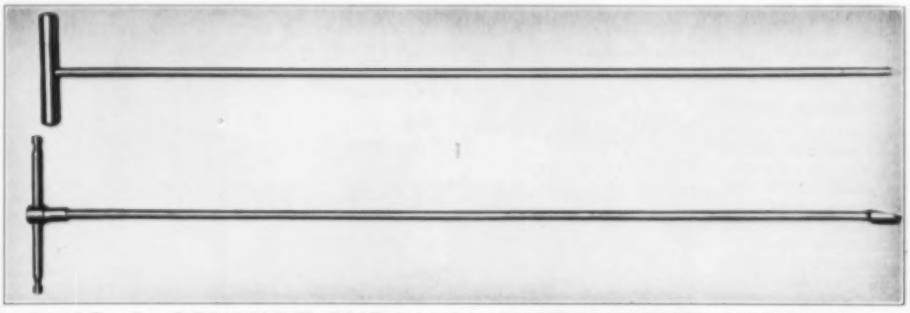

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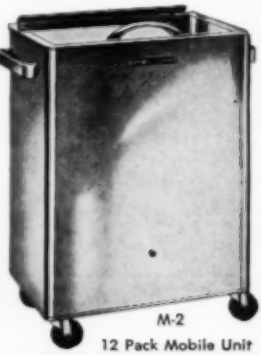

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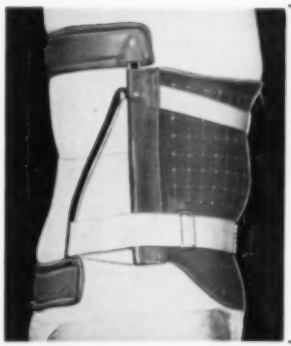

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